19343

Sam.l Lewis M.D.

COLLEGE OF PHYSICIANS PHILADELPHIA.

TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY

OF PHILADELPHIA.

VOLUME EIGHTH.

CONTAINING THE REPORT OF THE PROCEEDINGS FROM SEPTEMBER, 1877, TO JULY, 1878.

EDITED BY

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LECTURER ON HISTOLOGY IN THE UNIVERSITY OF PENNSYLVANIA; RECORDER OF THE SOCIETY.

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Former Presidents.

SAMUEL D. GROSS, M.D., LL.D. Oxon., elected 1857.
RENÉ LA ROCHE, M.D., elected 1858.
ALFRED STILLÉ, M.D., elected 1859, '61, '62 and '63.
EDWARD HARTSHORNE, M.D., elected 1863.
J. M. DA COSTA, M.D., elected 1864, '65 and '66.
JOHN H. PACKARD, M.D., elected 1867 and '68.
S. WEIR MITCHELL, M.D., elected 1869.
JOHN ASHHURST, Jr., M.D., elected 1870.
JAMES H. HUTCHINSON, M.D., elected 1871 and '72.
WILLIAM PEPPER, M.D., elected 1873.
H. LENOX HODGE, M.D., elected 1876.

OFFICERS AND COMMITTEES

OF THE

Pathological Society of Philadelphia.

President.

H. LENOX HODGE, M.D.,

ELECTED OCTOBER, 1876, FOR THREE YEARS.

ELECTED AT THE ANNUAL MEETING, OCTOBER, 1878.

Vice-Presidents.

W. F. NORRIS, M.D.,

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JOHN GUITÉRAS, M.D., CARL SEILER, M.D.,
H. F. FORMAD, M.D.

LIST OF MEMBERS.

Members marked N. R. are non-resident.
Members marked O. M. are of those originating the Society.

Elected

1857 Agnew, D. Hayes, Professor of Surgery in the University of Pennsylvania, 1611 Chestnut Street. (O. M.)
1871 Alison, Robert H., 250 South Seventeenth Street.
1864 Allen, Harrison, Professor of Physiology in the University of Pennsylvania, 117 South Twentieth Street.
1869 Allis, Oscar H., one of the Surgeons to the Presbyterian Hospital, 1604 Spruce Street.
1865 Andrews, Thomas H., 1117 Spruce Street.
1871 Ashbridge, William, Surgeon to the German Hospital, 1702 Chestnut Street.
1867 Ashhurst, Francis, Mount Holly, N. J. (N. R.)
1861 Ashhurst, John, Jr., one of the Surgeons to the Episcopal Hospital, 2000 West De Lancey Place.
1863 Ashhurst, Samuel, 1423 Walnut Street.
1871 Atlee, Washington L., Jr.*

1866 Bache, T. Hewson, 233 South Thirteenth Street.
1878 Baer, B. F., 1932 Bainbridge Street.
1876 Barr, J. W., 804 South Tenth Street.
1867 Bartles, William H., Insane Department Pennsylvania Hospital, West Philadelphia. (N. R.)
1869 Barton, J. M., 1344 Spruce Street.
1873 Beecher, A. C. W., 925 Walnut Street.
1870 Bell, J. R. F.*
1871 Bennett, Wm. H., 332 South Fifteenth Street.
1863 Benton, C. H. (N. R.)
1875 Bernardy, Eugene P., 221 South Seventeenth Street.
1870 Bertolet, Robert Morris. (N. R.)
1871 Betts, Thomas, Branchtown, Pa. (N. R.)
1860 Bishop, C. S., died.
1866 Black, J. J., New Castle, Delaware. (N. R.)
1863 Boardman, C. H., St. Paul, Minnesota. (N. R.)

Elected

1857 Boker, Charles S., one of the Surgeons to St. Joseph's Hospital, 1622 Chestnut Street. (O. M.)

1868 Bolles, Lucius S., died August 15th, 1873.

1877 Bradford, T. Hewson, 1905 Pine Street.

1873 Bray, Daniel, 1619 Vine Street.

1876 Brewster, W. B.*

1857 Brinton, John H., one of the Surgeons to the Philadelphia Hospital, 1423 Spruce Street. (O. M.)

1874 Brubaker, A. P., 217 North Thirteenth Street.

1874 Bruen, Edward T., one of the Visiting Physicians to the Philadelphia Hospital, 1531 Chestnut Street.

1871 Buck, William Penn, resigned October 23d, 1873.

1867 Burnett, C. H., Aural Surgeon to the Presbyterian Hospital, 127 South Eighteenth Street.

1872 Cathcart, James H., died March 5th, 1874.

1871 Cheston, C. Morris, West River, Maryland. (N. R.)

1865 Cheston, D. Murray, one of the Physicians to the Children's Hospital, 1839 Chestnut Street.

1871 Clark, Leonardo S., 1505 Girard Avenue.

1866 Cleemann, Richard A., one of the Physicians to St. Mary's Hospital, 340 South Twenty-first Street.

1872 Cohen, J. Solis, Lecturer on Laryngoscopy and Diseases of the Throat and Chest in the Jefferson Medical College, 1431 Walnut Street.

1877 Collins, James, 538 Marshall Street.

1869 Corbit, William B., Washington, D. C. (N. R.)

1877 Cruice, R. B., 731 North Seventeenth Street.

1871 Curtin, Roland G., Lecturer on Physical Diagnosis in the University of Pennsylvania, 332 South Seventeenth Street.

1857 Da Costa, J. M., Professor of Theory and Practice of Medicine in the Jefferson Medical College, 1700 Walnut Street. (O. M.)

1859 Darby, John T., New York City. (N. R.)

1876 Darlington, W. L., 146 North Twentieth Street.

1857 Darrach, James, 5094 Green Street, Germantown. (O. M.)

1868 Darrach, William, 42 Queen Street, Germantown.

1870 Deal, Lemuel J.*

1877 Deaver, R. W., 4613 Germantown Avenue.

1877 Drysdale, T. M., northeast corner Sixteenth and Arch Streets.

1866 Duer, Edward L., one of the Obstetricians to the Philadelphia Hospital, 1704 Arch Street.

1870 Duhring, L. A., Clinical Professor of Dermatology in the University of Pennsylvania, 1416 Spruce Street.

1876 Dulles, C. W., 220 South Fortieth Street.

Elected

1860 Dunton, W. R., 5059 Germantown Avenue, Germantown.
1863 Dyer, Ezra, Pittsburg, Pennsylvania. (N. R.)

1875 Engel, Hugo, northeast corner Fifth and Wood Streets.
1876 Eskridge, J. T., 1622 North Sixteenth Street.

1857 Fischer, Emil, southeast corner Sixth and Brown Streets. (O. M.)
1863 Fish, Augustine H., died August 3d, 1872.
1876 Fisher, Henry, 919 Walnut Street.
1857 Forbes, William S., one of the Surgeons to the Episcopal Hospital, 1405 Locust Street. (O. M.)
1869 Ford, William H., 1622 Summer Street.
1878 Formad, H. F., 105 South Thirty-fourth Street.
1871 Fox, Charles, resigned December 10th, 1874.

1868 Garretson, James E., 1537 Chestnut Street.
1871 Gerhard, George S., Ardmore. (N. R.)
1870 Getchell, Frank H., Clinical Lecturer on Diseases of Women in the Jefferson Medical College, 1432 Spruce Street.
1877 Gilliard, Louis, died Nov. 1878.
1877 Goldsborough, C. B. (N. R.)
1866 Goodell, William, Clinical Professor of Diseases of Women in the University of Pennsylvania, Preston Retreat, Twentieth and Hamilton Streets.
1868 Goodman, H. Earnest, one of the Surgeons to the Orthopædic Hospital, 1427 Chestnut Street.
1876 Gorgas, S. R.*
1868 Graham, James, resigned January 9th, 1879.
1867 Grier, Matthew J., 1531 Spruce Street.
1868 Gross, Ferdinand H., one of the Surgeons to the German Hospital, 608 North Sixth Street.
1857 Gross, Samuel D., Professor of Surgery in the Jefferson Medical College, southeast corner of Eleventh and Walnut Streets. (O. M.)
1858 Gross, Samuel W., one of the Visiting Surgeons to the Philadelphia Hospital, 1112 Walnut Street.
1869 Grove, John H., one of the Surgeons to St. Mary's Hospital, 1330 Arch Street.
1872 Gruel, Theodore H. E., 612 Spruce Street.
1874 Guitéras, John, one of the Visiting Physicians to the Philadelphia Hospital, 1912 Vine Street.

1871 Hale, George J.*
1857 Hall, A. Douglass, resigned October 9th, 1873. (O. M.)
1875 Hand, Frank C., 830 Pine Street.
1866 Hare, Horace Binney, died March 21st, 1879.

Elected

1868 Hargadine, R. W. (N. R.)

1859 Harlan, George C., one of the Surgeons to Wills Ophthalmic Hospital, 1806 Chestnut Street.

1874 Harris, Charles M., 715 South Twenty-third Street.

1858 Harris. Robert P., resigned January 9th, 1873.

1857 Hartshorne, Edward, 331 South Broad Street. (O. M.)

1859 Hartshorne, Henry, resigned October 12th, 1864.

1867 Hassler, Ferdinand A., resigned February 9th, 1871.

1869 Hatfield, Nathan L., one of the Visiting Surgeons to the Philadelphia Hospital, 501 Franklin Street.

1868 Hayes, I. Minis, 1607 Locust Street.

1879 Hazlehurst, S. F., one of the Assistant Physicians to the Children's Hospital, 1808 Pine Street.

1873 Hearn, Joseph, 324 Catharine Street.

1870 Henry, Frederick P., one of the Physicians to the Episcopal Hospital, 635 Spruce Street.

1866 Herbert, Theodore. (N. R.)

1879 Hess, Robert, Resident Physician Pennsylvania Hospital for Insane.

1857 Hewson, Addinell, southwest corner of Twenty-first and Walnut Streets. (O. M.)

1874 Hickman, N. B., 326 South Sixteenth Street.

1858 Hodge, H. Lenox, one of the Surgeons to the Presbyterian Hospital, 506 South Broad Street.

1871 Hoffman, Washington Atlee, died September 20th, 1874.

1860 Hopkins, Henry St. George. (N. R.)

1876 Hopkins, W. B., 2018 Spruce Street.

1877 Horwitz, Theodore, died December 12th, 1877.

1870 Houston, James P. S., Savannah, Georgia. (N. R.)

1866 Howe, Herbert M., resigned February 28th, 1878.

1859 Hoyt, William D. (N. R.)

1857 Humphries, George H., New York City. (O. M., N. R.)

1868 Hunter, Charles T., Demonstrator of Surgery in the University of Pennsylvania, 1905 Walnut Street.

1870 Hutchins, E. R. (N. R.)

1858 Hutchinson, James H., one of the Physicians to the Pennsylvania Hospital, 2019 Walnut Street.

1866 Ingham, James V., one of the Obstetricians to the State Hospital for Women, 1342 Spruce Street.

1876 Ingram, T. D., 1601 Vine Street.

1874 Jameson, Edward, St. Louis, Mo. (N. R.)

1866 Jenks, William F., Surgeon to the State Hospital for Women. (N.R.)

1875 Johnson, Russell H., one of the Assistant Physicians to the Children's Hospital, 2127 Spruce Street.

*

Elected

1859 Kane, John K., Wilmington, Delaware. (N. R.)
1874 Keating, John M., one of the Visiting Physicians to the Philadelphia Hospital, northwest corner Twenty-second and Locust Streets.
1857 Keating, William V. (O. M.*)
1866 Keen, William W., Professor of Artistic Anatomy, Pennsylvania Academy of Fine Arts, 1729 Chestnut Street.
1857 Keller, William, resigned January 28th, 1875. (O. M.)
1875 Kelly, Joseph V., 4257 Main Street, Manayunk.
1858 King, William, United States Navy. (N. R.)
1869 Knight, Samuel R., Superintendent of the Episcopal Hospital.

1867 Lambdin, Alfred C.*
1871 Landis, Henry G., Niles, Ohio. (N. R.)
1872 La Roche, C. Percy, Nice, France. (N. R.)
1857 La Roche, René. (O. M.) Died December 9th, 1872.
1870 Leach, Alonzo L., 2211 Spruce Street.
1869 Leaman, Henry, 1031 Vine Street.
1860 Lee, Charles C., New York City. (N. R.)
1861 Leedom, John M., Pulaski Avenue, Germantown.
1857 Levick, James J., resigned October 13th, 1861. (O. M.)
1857 Levis, Samuel, 1330 Spruce Street. (O. M.)
1868 Lewis, Francis W., 2016 Spruce Street.
1869 Lewis, Fred. W., died 1873.
1875 Lewis, Morris J., one of the Assistant Physicians to the Children's Hospital, 1216 Walnut Street.
1877 Little, William S., 256 South Sixteenth Street.
1859 Livezy, Edward,* died 1876.
1876 Loder, P. E., 517 South Eighth Street.
1873 Longenecker, Jerome.*
1870 Longstreth, Morris, Pathologist to the Pennsylvania Hospital, 333 South Twelfth Street.
1870 Loughlin, J. Eneu.*

1874 Mann, Charles H. (N. R.)
1867 Markoe, James, 2217 St. James Place.
1868 Martin, George. (N. R.)
1874 Matteson, Charles C., died.
1865 Maury, Frank F., one of the Surgeons to the Philadelphia Hospital, 1218 Walnut Street.
1870 Maxwell, J. Gordon,* died.
1866 McArthur, John A., 406 South Broad Street.
1861 McCall, C. A., resigned.
1868 McClure, W. Wallace, resigned September 10th, 1878.
1873 McCoy, A. W., Pittsburg, Pa. (N. R.)

Elected

1864 Mears, J. Ewing, one of the Surgeons to St. Mary's Hospital, 1429 Walnut Street.

1873 Meigs, Arthur V., one of the Assistant Physicians to the Children's Hospital, 1322 Walnut Street.

1857 Meigs, John Forsyth, one of the Physicians to the Pennsylvania Hospital, 1208 Walnut Street. (O. M.)

1874 Miller, C. K. I., resigned December 28th, 1876, died 1878.

1878 Mills, C. K., 1502 Columbia Avenue.

1857 Mitchell, S. Weir, one of the Physicians to the Hospital for Nervous Diseases, 1524 Walnut Street. (O. M.)

1876 Montgomery, E. C., southeast corner of Twentieth and Jefferson Streets.

1857 Morehouse, George R., 227 South Ninth Street.

1857 Morton, Thomas George. (O. M.*)

1859 Moss, William, Chestnut Avenue, Chestnut Hill.

1871 Muhlenberg, Frank, 1909 Chestnut Street.

1869 Müller, August F., 4544 Germantown Avenue.

1873 Musser, Milton B., Fortieth and Locust Streets.

1869 Mustin, J. Burton, died 1871.

1869 Nancrede, Charles B., one of the Surgeons to the Episcopal Hospital, 2109 Pine Street.

1879 Neff, J. S., Resident Physician Pennsylvania Hospital.

1872 Newsham, Stanley P., 1507 South Fifth Street.

1868 Norris, Herbert, 313 South Eighteenth Street.

1868 Norris, Isaac, resigned November 12th, 1874.

1869 Norris, J. C., resigned February 10th, 1876.

1861 Norris, William F., Clinical Professor of Ophthalmology in the University of Pennsylvania, 1526 Locust Street.

1875 O'Hara, Michael, 31 South Sixteenth Street.

1874 Osgood, Hamilton. (N. R.)

1857 Packard, John H., one of the Surgeons to the Episcopal Hospital, 1928 Spruce Street. (O. M.)

1868 Pancoast, William H., Professor of Anatomy in the Jefferson Medical College, 1100 Walnut Street.

1873 Parish, William H., 324 South Seventeenth Street.

1875 Parks, Edward L. (N. R.)

1867 Parry, John S., died 1876.

1870 Paul, Comegys, Resident Physician Eastern Penitentiary.

1875 Paul, James, 1608 Walnut Street.

1857 Penrose, R. A. F. (O. M.*)

1865 Pepper, George, died September 14th, 1872.

1865 Pepper, William, Professor of Clinical Medicine in the University of Pennsylvania, 1811 Spruce Street.

Elected

1879 Perkins, F. M., 1428 Pine Street.

1870 Porter, William G., Jr., one of the Surgeons to the Presbyterian Hospital, 314 South Eleventh Street.

1878 Raub, J. A., 927 North Tenth Street.

1874 Reed, Henry B., one of the Assistant Surgeons to the Episcopal Hospital, 2300 De Lancey Place.

1859 Reed, Thomas B., one of the Surgeons to the Presbyterian Hospital, 1427 Walnut Street.

1871 Rex, George A., 2118 Pine Street.

1869 Rex, Oliver P.*

1877 Reynolds, A. S., 1801 Poplar Street.

1864 Rhoads, Edward, died January 16th, 1871.

1878 Rich, T. C., 622 South Sixteenth Street.

1869 Richardson, Elliott, Lecturer on Practical Obstetrics in the University of Pennsylvania, 737 Spruce Street.

1863 Richardson, Joseph G., one of the Visiting Physicians to the Presbyterian Hospital, 1835 Chestnut Street.

1857 Richardson, T. G., New Orleans, La. (O. M., N. R.)

1876 Risley, S. D., Lecturer on Ophthalmoscopy in the University of Pennsylvania, 1630 Walnut Street.

1868 Ritz, Charles M. (N. R.)

1876 Roberts, J. B., 1118 Arch Street.

1876 Roland, O. (N. R.)

1874 Ronaldson, William D., 4017 Locust Street.

1877 Rudderow, B. J., 527 North Nineteenth Street.

1867 Santee, Eugene J., 532 North Sixth Street.

1863 Savery, William, resigned February 11th, 1869.

1869 Schell, H. S., 1802 Chestnut Street.

1874 Seiler, Carl, 1608 Pine Street.

1870 Seyfert, Theodore H., resigned December 27th, 1877.

1870 Shaffner, Charles, 23 South Sixteenth Street.

1876 Shakespeare, E. O., Lecturer on Refraction and Accommodation and Operative Ophthalmic Surgery in the University of Pennsylvania, 1336 Spruce Street.

1869 Shapleigh, Elisha B., 658 North Eighth Street.

1868 Sherwood, Thomas H.*

1878 Shoemaker, J. V., 1031 Walnut Street.

1871 Simes, J. H. C., Lecturer on Histology in the University of Pennsylvania, 2033 Chestnut Street.

1868 Sinkler, Wharton, one of the Physicians to the Hospital for Nervous Diseases, 1534 Pine Street.

1858 Smith, A. H., Lecturer on Obstetrics at the Philadelphia Lying-in Charity, 1419 Walnut Street.

Elected

1863 Smith, E. A., 126 South Eighteenth Street.
1859 Smith, F. G., resigned November 13th, 1873. Died 1878.
1876 Smith, R. M., 25 South Sixteenth Street.
1875 Smith, Stanley. (N. R.)
1876 Smyth, F. G., 1212 South Tenth Street.
1871 Sparks, George W.*
1866 Spooner, Edward A.*
1871 Starr, Louis, one of the Physicians to the Episcopal Hospital, 1417 Spruce Street.
1857 Stillé, Alfred, Professor of Theory and Practice of Medicine in the University of Pennsylvania, 3900 Spruce Street. (O. M.)
1870 Strawbridge, George, resigned June 10th, 1875.
1869 Stryker, Samuel S., one of the Obstetricians to the Philadelphia Hospital, 3709 Walnut Street.

1879 Taylor, J. M., 2212 Fitzwater Street.
1866 Thomas, Charles H., 1807 Chestnut Street.
1868 Thomson, William, one of the Surgeons to Wills Ophthalmic Hospital, 1502 Locust Street.
1870 Tinkham, J. H., United States Navy. (N. R.)
1866 Townsend, George D. (N. R.)
1868 Townsend, R. M., died.
1864 Turner, A. P. (N. R.)
1863 Tutt, Charles Pendleton, died May 11th, 1866.
1863 Tyson, James, Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania, 1506 Spruce Street.

1871 Valdivieso, R. (N. R.)
1870 Van Harlingen, Arthur, Chief of the Dispensary for Diseases of the Skin, Hospital of the University of Pennsylvania, 129 South Fifteenth Street.

1874 Warder, Wm. H., one of the Obstetricians to the Philadelphia Hospital, 810 North Broad Street.
1867 Watson, E. W., 146 North Twentieth Street.
1866 Weightman, John F., resigned April 27th, 1871.
1876 Weir, A. H., 130 South Fifteenth Street.
1875 West, Franklin, died.
1875 Whelen, Alfred, Senior Assistant Physician to the Philadelphia Lying-in Charity, 1814 Locust Street.
1873 White, J. William, one of the Visiting Surgeons to the Philadelphia Hospital, 222 South Sixteenth Street.
1868 Willard, De Forrest, Lecturer on Orthopædic Surgery in the University of Pennsylvania, 1626 Chestnut Street.
1865 Williams, Horace, one of the Obstetricians to the State Hospital for Women, 1717 Pine Street.

Elected

1875 Williamson, Jesse, one of the Surgeons to Howard Hospital, 1711 Pine Street.
1867 Wilson, Ellwood, resigned.
1869 Wilson, James C., one of the Visiting Physicians to the Philadelphia Hospital, 1437 Walnut Street.
1867 Wilson, James F., 1010 Race Street.
1869 Wilson, J. H. (N. R.)
1873 Winslow, W. H., resigned December 28th, 1876.
1864 Wistar, Thomas, resigned February 13th, 1867.
1875 Wolford, W. S., 133 South Thirteenth Street.
1863 Wood, Horatio C., Jr.*
1865 Woods, D. Flavel, one of the Physicians to the Presbyterian Hospital, 151 North Fifteenth Street.
1857 Woodward, J. J., United States Army, Washington, D.C. (O.M., N.R.)
1872 Worthington, David J., 116 South Fifteenth Street.

1876 Zeigler, G. W. (N. R.)

Members marked * have forfeited their membership.

CORRESPONDING MEMBERS.

1866 Bumstead, Freeman J., Professor of Venereal Diseases in the College of Physicians and Surgeons, New York.
1859 Clark, Alonzo, Professor of Theory and Practice of Medicine in the College of Physicians and Surgeons, New York.
1859 Dalton, J. C., Professor of Physiology in the College of Physicians and Surgeons, New York.
1860 Ellis, Calvin, Professor of Theory and Practice of Medicine in the Medical Department of Harvard University, Boston, Massachusetts.
1858 Flint, Austin, Sr., Professor of Theory and Practice of Medicine in Bellevue Hospital Medical College, New York.
1858 Hammond, William A., Member of the National Academy of Sciences, New York.
1860 Isaacs, Charles E., Brooklyn. Died June 16th, 1860, æt. 48.
1878 Jackson, J. B. S., Boston. Died 1879.
1860 Reeves, James E., Wheeling, West Virginia.
1861 Rokitansky, Carl. Died 1878.
1860 Watson, John, New York. Died June 3d, 1863, æt. 56.
1859 Worthington, Wilmer, West Chester, Pennsylvania. Died 1873.

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REPORT.

I.—THE OSSEOUS SYSTEM.

1. *Medullary sarcoma.*

By Dr. F. H. Gross, for Dr. James Collins.

The specimen of medullary sarcoma which it is my privilege to exhibit to the Society for my colleague Dr. James Collins was removed from a young patient at the German Hospital, by amputation of the left arm above the condyles of the humerus.

From the notes kindly furnished me by Dr. C., who regrets that they are somewhat meagre, I prepare the following history of the case:

Samuel S. R., aged 8 years, of healthy American parentage, sustained in the latter part of November, 1876 (nearly eleven months ago), an injury to the forearm while in bed. He was treated for some time by a neighboring apothecary with lotions and liniments, but without any improvement.

The doctor's attention was first called to the case early in February last, when he found a tumor of the size of an egg occupying the middle of the ulna, and which seemed to clasp the ends of an ununited fracture of that bone. This enlargement was held to be an excessive mass of vitiated callus. As the surroundings of the little patient led to the belief that he could not be properly treated at home, Dr. C. strongly urged the lad's parents, who were poor, to send him to a hospital, in the hope that a useful hand might be preserved. But this advice was not then acted upon.

A few weeks later it was noticed that the growth was rapidly increasing in size, that the superficial veins of the forearm were becoming very prominent, that the part was severely painful, and that the general health of the patient was becoming somewhat impaired. There was, however, nowhere in the limb any evidence of glandular enlargement.

It was now held to be a sarcoma, and, as the disease was becoming constantly more aggravated, the patient was finally, on the 3d of October, after the repeated advice of Dr. Collins, admitted into the German Hospital, where, two days later, that gentleman amputated the arm above the elbow, in the presence of several members of the hospital staff and the resident physicians.

Up to this time, the sixth day after the operation, the patient continues to do very well.

The amputated portion of the humerus, and the hand, which were not involved, have been disarticulated from the diseased forearm. The tumor, which measured in its greatest circumference fourteen inches and in its length seven inches, has been bisected longitudinally. The portion here presented has been preserved in a solution of hydrate of chloral, and the other half is being prepared for microscopic study.

Dr. C. expresses his indebtedness for the note, which I copy below, from Dr. S. W. Gross, who saw the case and examined the specimen:

"The section of the tumor was six inches long by four and a half inches broad at its widest part. The growth had developed at the seat of fracture of the ulna, which was about midway of the bone, and was evidently periosteal in its origin, although fully one-half grew from the outer surface of the periosteum. At the point of fracture were numerous flattened, radiating spiculæ of bone. The upper and lower epiphyses of the ulna were not involved.

"The whole appearances of the tumor were those of encephaloid or medullary sarcoma. The brain-like tissue, more especially the central portions, was very soft, and easily broken down. It was the seat, here and there, of calcareous degeneration and cystic transformation, and pervaded, particularly at the lowermost portion, by injected vessels and hemorrhagic deposits.

"From the history of the case, and from the gross appearances of the tumor, I have no hesitation in pronouncing it a small round-celled sarcoma."

October 11*th*, 1877.

Report of the Committee on Morbid Growths:

"The specimen presented by Dr. F. H. Gross, and referred to the Committee on Morbid Growths for examination, presents the structural arrangement and elements of a round-celled sarcoma. A thin section demonstrates the new formation to consist of round, lymphoid cells, with scarcely any visible intercellular substance. The blood-vessels are seen to be simple channels through the cells of the growth." *November* 8*th*, 1877.

2. *Dislocation of cervical vertebræ, with injury of the spinal cord.*

Presented by Dr. M. Longstreth.

Chas. K., æt. 37, German, admitted to the Pennsylvania Hospital October 18th, 1875. Two days previously he had fallen backwards off a fence on which he was sitting, three or four feet to the ground, striking the back about the region of the shoulders. On admission, there was complete paralysis of motion in the legs; right arm could be moved a little, but not the left. Head can be turned only slightly to the right. No sensation below the level of the nipples on the trunk and legs, none on the arms below the deltoid region. Diaphragm moves freely. Retention of urine. No movement of the bowels. Priapism. Perfectly conscious and rational.

In the evening of the 18th, respiration became difficult, 48 per minute; pulse, 116; temperature, 101.5° Fahr.

The difficulty of respiration gradually increased, shortly before death becoming entirely intercostal in its type. Death on the third day.

For the clinical notes of the case, taken from the hospital records, I am indebted to Dr. J. B. Roberts.

The fifth cervical vertebra is separated from the cartilage on its under surface, and slides forward half the width of its body on the sixth vertebra. All the ligaments are torn, excepting a part of anterior common ligament, and those of the transverse processes, while the vertebræ themselves appear uninjured at this point. The spinous processes of the first and second dorsal vertebræ are fractured at a point about half-way in their length.

The spinal cord makes a bend and is compressed at the point of dislocation; the dura mater, as well as the ruptured cartilage and ligaments of the vertebra, is stained red. At the *autopsy* there was found considerable blood effused into the muscular tissue of the neck localized around the injury. The dura mater is reddened also at the part situated in the body of second dorsal. At both of these points the dura is slightly distended; at the upper point it is somewhat broadened and flattened by pressure, whilst at the lower one it is rounded. On laying open the dura along its posterior surface, no adhesions were found between it and the arachnoid; the arachnoid was normal and transparent, showing no evidence of hemorrhage, and no adhesion to the pia mater.

At the seat of the injuries the pia mater and cord show a darker color, are slightly enlarged; the cord is very much softened, giving a feeling of fluctuation, but, on incision, it is not found diffluent, although the nervous

tissue wells up through the cut surface of the membrane. The softening is more marked at the upper injury.

The length of the cord between these two places is decidedly less firm than normal, but otherwise shows no marked change.

Microscopically, the nervous tissue taken from the softened cord shows abundance of blood-coloring matters, with numerous broken tubules, quantities of granular detritus, and a very few large granular corpuscles.

January 10*th*, 1878.

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3. *Old inflammatory changes and anchylosis of cervical and dorsal vertebræ.*

Presented by Dr. Morris Longstreth.

Richard E., æt. 50, colored, admitted to the Pennsylvania Hospital, November 2d, 1873, having fallen down-stairs at 3 A.M. of that day. He lay there some hours, unable to move or call for help; was brought to the ward at 6 A.M., unable to articulate, arms and legs paralyzed; the neck was prominent in front, and a depression was felt at about the seventh cervical spine. In the afternoon he could talk without difficulty; there was paralysis of motion and sensation of left hand and forearm, but he had regained the power of motion, and sensation was perfect over the rest of the body. He could place either hand on his head, but moved them hesitatingly. The head moved a little to the left side, but not to the right. Complained of discomfort, but no severe pain. He required the use of the catheter. He was found dead at midnight, having continued in the same condition until that time. There was some evidence which led us to think that a sudden movement of the body caused a dislocation of the fracture. There was no history obtainable of previous disease affecting the neck or throat, and no evidence of syphilitic infection.

The clinical notes of the case were taken by Dr. Woodbury from the hospital records.

The specimen shows disease of all the cervical vertebræ, most markedly on their bodies and articular processes. The atlas is not shown in the preparation, but examination of it at the *autopsy* found it free from disease. In the removal of the vertebræ from the body, the saw-cut removed the odontoid process, which was also normal. The axis and the third vertebræ are fused into one mass, showing their parts, however, distinctly. The

anchylosis affects not only the bodies at the intervertebral discs, and the articulation at the transverse processes, but also the back parts of their arches at the base of the spinous processes.

The lower part of the third vertebra has the lower part of its body, which when normal projects downward and forward to meet the fourth, very much spread out, and shelved forward more than a half-inch, and this part is very uneven and jagged with small spiculæ of bone. It would seem as though part of its body had been destroyed by caries. The junction of the third and fourth vertebræ gives the appearance on their anterior surface of a backward dislocation of the former, but the interior of the spinal canal disproves the occurrence of this condition.

The body of the fourth vertebra is increased in size in all directions; its upper border projects upward, and has a ragged edge slightly overlapping the remains of the intervertebral cartilage, as well as the vertebra above it; its central part is more prominent than normal; its lower border (epiphyseal plate) shows a line of separation perforated with large canals. This vertebra was pretty firmly united with the one below it, but there still remained a thin layer of unossified cartilage between them; their line of junction is very irregular and jagged.

The fifth and sixth vertebræ are firmly anchylosed throughout, showing as the only openings between them the two intervertebral foramina, and a small fissure at the base of their spinous processes; their bodies are considerably changed in form, broadened and irregular laterally, with anteriorly a smooth concave surface, perforated by numerous openings, but showing no trace of the normal line of separation; their laminæ are broadened and thickened, and their transverse processes rounded and massive; their upper and lower terminations are very irregular, the lower one especially, which overlaps the seventh vertebra considerably on its right side.

The seventh vertebra has its body distorted, its anterior surface roughened and irregular, the edges projecting and jagged. The intervertebral cartilage between it and the first dorsal is normal, but the first dorsal has suffered to the same degree as the seventh cervical; both of them except in their bodies appear nearly normal.

The examination of the spinal cord was very imperfectly carried out, it being impossible to remove it except in fragments, without destroying the bones as a specimen, owing to the firmer connection of the dura mater in the spinal canal than usual.

The dura mater appeared, as seen through the opening between the separated and the fractured vertebræ, uninjured, but having a red color

from blood-staining. The cord was softened, and there was considerable hemorrhage at the seat of injury.

The fracture and the probably subsequent dislocation were at the junction of the sixth and seventh cervical vertebræ.

Remarks.—The condition of the bone was the result of disease which long since had healed, although no history could be obtained indicating the time of its occurrence or its nature: in fact, there was no knowledge of the existence of any such condition until after the *autopsy* was made. While it is probable that the patient himself could have furnished the information, had he been in a condition to communicate it, his friends knew nothing of it.

When death results *suddenly* from injury to the cervical portion of the spinal cord, I think it becomes a question whether it is not in such cases due to cardiac paralysis. I have no knowledge, however, of the precise mode of death from such injuries, and it would require a large number of *post-mortem* examinations to determine this question from the state of contraction of the ventricular cavities, which is so expressive in such points. If the death were due to a cardiac paralysis, coming suddenly and of purely nervous origin, its origin, I think, must be traced to injury of the cord, or the spinal nerve fibres communicating with cervical sympathetic ganglia, or their interganglionic cords from which arise the cardiac nerves passing to the special plexuses, or ganglia of the heart.

Dr. John Ashhurst, Jr., said, in regard to the cause of death in cases of dislocation of the vertebræ, that if the lesions were above the fourth cervical vertebra, instant death would result from implication of the phrenic nerve. When lower down, death was ordinarily due to paralysis of the diaphragm, caused not only by extension of spinal softening in an upward direction, but mechanically also by distention of the intestines with gas, from paralysis of the abdominal muscles. In answer to a question as to the frequency of dislocation of the vertebræ without fracture, Dr. A. said that it had been thought to be very rare by the older writers, but that later authors had mentioned it as occurring more frequently than was formerly supposed. He had himself never seen a case that merited the name of pure dislocation, and he believed there was generally some bone lesion present. Some years ago he had proposed the name *diastasis* for these cases, as there was a separation rather than a luxation such as occurs in the joints of the extremities. The lesion was most frequently met with in the cervical region, next in the lumbar, and least often in the dorsal spine, where the ribs act as splints in preventing displacement.

January 10*th*, 1878.

4. *Case of fracture of the femur in an infant.*

Presented by Dr. O. H. Allis.

J. K., 18 months of age. About two weeks before its death, the mother noticed that the child cried violently when she attempted to put on the right shoe. A week later I saw the child once, and examined the ankle, knee, and hip, and did not detect any unusual symptoms. The child cried, but not worse than when I examined the left limb or the abdomen. The child had a slight cough, was teething, and its urine was dark and turbid. There was no physician in attendance, but just before its death a neighboring physician was called in.

Autopsy, twenty-four hours after death.—The viscera in the abdominal and thoracic cavities gave no clue to the cause of death. The mesenteric glands were considerably enlarged, and the ribs at their sternal extremities were markedly rounded and enlarged.

On examining the right femur I found a fracture of the neck, with loss of substance, and also a separation of the condyloid epiphysis with the periosteum, separated from the shaft nearly to the middle of the bone. The extremity of the shaft was injected, and there was evidence that blood had been effused beneath the periosteum. There were no traces of inflammatory change, and it is therefore not easy to state at what time the fracture of the neck or the separation of the epiphysis took place. There was, however, abundant evidence that it took place prior to death.

On examining the left limb, the neck of the femur was found to be intact, but a partial separation of the condyloid epiphysis was detected, without, however, any evidence that blood had been effused.

The mother thinks that the origin of the difficulty may be traced to a traumatic injury, viz., to an older sister (one three years old) sitting upon the body and thighs of the babe about two weeks before its death. Had this been the origin of the difficulty, there could scarcely be an entire absence of effort on the part of nature to repair the injury.

March 14*th*, 1878.

II.—THE DIGESTIVE APPARATUS.

1. *Cancer of the stomach.*

By Dr. E. T. Bruen.

M. McD., æt. 50, Irish, laborer, was admitted to the Philadelphia Hospital, August 8th, 1877. When admitted he stated that his family were all healthy people, and that he himself had enjoyed exceptional immunity from sickness until three years ago. At that time he noticed that whenever he ate articles of food which were difficult to digest, he would vomit two or three hours after eating. He had other dyspeptic symptoms at intervals, but, as he was careful about his diet, he did not vomit as much for a year previous to the date of this history.

About three months ago he commenced to be troubled with constipation and a feeling of soreness over the abdomen.

On admission, he was noticed to be emaciated, and his face had a pale cachectic hue. The lungs were healthy; a systolic murmur was heard at the base of the heart transmitted towards the epigastrium, and the veins of the neck were observed to pulsate violently. It appeared that the murmur was due to some tricuspid regurgitation.

On examining the abdomen, a tumor was felt in the epigastrium, extending three inches to the left of the median line and about four inches from the edge of the ribs on the left side of the chest. The tumor appeared to be superficial, but by depressing the abdominal wall it was found to extend deeply into the cavity of the abdomen towards the back. The tumor pulsated with the cardiac action, and on auscultation a murmur could be detected, which was increased by pushing the stethoscope deeply into the centre of the pulsating area, which was about two (2) inches in circumference; there was no thrill, and the pulsation disappeared when the patient assumed a position on the hands and knees. But little dulness could be detected on percussion over the tumor, and that only when the abdominal walls were deeply depressed. A slight murmur could be heard along the course of the aorta by auscultation over the back. The tumor was hard, no nodulation could be felt, but the rigidity of the abdominal muscles prevented further localization of the mass.

The temperature during the first four weeks after his admission ranged between 100° F. and 102° F. No albumen could be found in the urine. He complained bitterly of severe dull pain in the epigastrum and lumbar region, the pain in the back being especially severe. He was constipated most of the time he was under my care, but occasionally he had diarrhœa for a few days. No vomiting occurred while he was in the ward, but his diet was restricted, and principally liquid.

He gradually emaciated, and died from exhaustion, October 9th, 1877. His temperature, however, was normal for several weeks before his death, and the tricuspid murmur had disappeared.

Post-mortem, twenty-four hours after death.—Extreme muscular rigidity. Examination of chest revealed heart and lungs to be healthy. Tricuspid and mitral valves normal.

On opening the abdomen, the stomach was found extremely dilated, occupying the left hypochondriac region. The hard mass felt during life was seen to be connected with the stomach. The transverse colon and pancreas were found adherent to the greater curvature of the stomach, which was the seat of cancerous disease, apparently scirrhus.

On opening the stomach along the greater curvature, a cancerous mass as large as an orange occupied the posterior wall and lesser curvature, extending from within two inches of the cardiac orifice, towards the pylorus, which was also the seat of cancer. The portion around the pylorus was hard and smooth, but on the posterior wall lobules extended from the mass into the cavity of the stomach. The surface of the large mass occupying the posterior wall was softened, and the mucous membrane covering the posterior wall of the stomach was very much softened. Portions of the mass were friable. On the anterior surface of the anterior wall of the stomach small nodular masses the size of peas were found scattered, but apparently in the line of the lymphatics. The liver and kidneys were healthy. The mesenteric glands were very generally enlarged to the size of large peas. The spleen was of normal size, but its capsule was the seat of calcareous degeneration, and a tendency to lobulation of the spleen was observed.

Remarks.—This tumor during life very often simulated aneurism, but was differentiated from it by the absence of murmur when the position was changed, and the absence of pulsation when the patient was on the hands and knees. The dyspeptic symptoms, however, the cachexia, emaciation, and constipation, pointed to cancer, and the location and superficial character of the tumor pointed in my mind to cancer of the walls of the stomach. I would call attention to the temperature as an unusual feature of the case. Wunderlich says that temperature above 100° points against

a diagnosis of cancer; though recently I noticed a case of cancer of the liver with a temperature above 100° F., rising at times to 103° F.

The *post-mortem* threw no light upon the cause of the tricuspid murmur.

It is interesting to note that although this tumor was really so deeply seated it appeared superficial, and the absence of dulness was explained by the dilated condition of the healthy portion of the stomach. It was thought at one time that enlargement of the left lobe of the liver would explain the tumor; but absence of dulness in the epigastrium set aside this theory. No microscopic examination has yet been made, owing to the recent date of death of the patient, but I would desire to have the specimen referred to the Committee on Morbid Growths for their consideration.

October 11*th*, 1877.

Report of the Committee on Morbid Growths:

"A microscopical examination of the new formation developed in the stomach, presented by Dr. Bruen, shows the globular, polyp-like elevations situated at the pyloric region to consist of villous prolongations, covered with columnar epithelial cells, growing in every direction. From such an arrangement of elements the growth may be considered a cylindrical epithelioma." *November* 8*th*, 1877.

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2. *Abscesses of the liver.*

By Dr. John Guitéras.

Patrick G., æt. 39, admitted to the Philadelphia Hospital October 9th, 1877. Has always been a healthy man. Never had renal or pulmonary disease. Never had rheumatism. No syphilitic history. The family history is good. Has been working along the Susquehanna River, near Harrisburg, this fall. Three weeks ago had a chill, with fever. The chills and fever continued daily, with night-sweats. He has lost some flesh. He arrived in the city to-day, and walked to the hospital. He is a stout man, and does not seem to be very sick. Is not very pale. The heart and lungs are healthy. Appetite poor. Bowels costive. Liver enlarged, extending from the fourth rib to half-inch below the margin of thorax. Posteriorly it extended up to half an inch below scapula. Complains of some pain over the liver. Tongue coated; no vomiting, nor has there been at any time. Cannot sleep at night; has fever. (See temperature.)

October 10*th*, 1877.—Fever continues. Quinine ordered. Nourishing liquid diet.

October 11*th.*—Pain over the liver, with tenderness, especially towards the epigastrium. Bowels constipated. Quinine continued. Ordered castor oil.

On the 12th, 13th, and 14th, no change. Quinine had not been taken; was ordered, producing slight change in the temperature. Complained but little of pain in the liver.

15*th.*—Bowels moved in the morning. Got up in absence of the nurse, and walked down-stairs to smoke a pipe. In the afternoon had profuse hemorrhages from the bowels. Hypodermic injections of ergotin arrested the hemorrhage; but active stimulation failed to bring about reaction. He died at six o'clock the next morning.

Post-mortem.—Lungs healthy. The right lung is adherent to the diaphragm. Heart healthy. Kidneys slightly fatty. Spleen normal. Small intestine healthy.

Large intestine congested. The solitary glands enlarged. At the cæcum there are some large, irregular, but very superficial ulcers, without any infiltration of the surrounding tissue. About six inches from the valve, an ulcer with an infiltrated base was found. It is a transverse ulcer, extending almost all the way around the intestine. Its surface is covered with a slough. The large intestine contains a large amount of blood.

The liver is uniformly enlarged, very pale, flabby, and decidedly fatty. An abscess about the size of a fœtal head occupies the convexity of the right lobe. It has reached near the capsule, but there are as yet no adhesions to the diaphragm. Two abscesses that would hold a middle-sized orange each are found in the line of the longitudinal fissure and in the left lobe, near the upper surface, anteriorly. There is also a smaller one in the right lobe.

I dissected the branches of the portal vein, and found leading to every one of the abscesses a large branch of this vein, which seemed to have melted in the suppurative process. These branches terminated abruptly, and were only separated from the pus by the so-called pyogenic membrane. These blood-vessels showed no evidence of phlebitis, neither could I find any embolus. The peritoneal covering at the seat of the intestinal ulcer was perfectly normal, and I could find no evidence of phlebitis there.

Temperature Record.—*October* 9*th.*—Evening, P. 96, resp. 25, temp. 100°.

October 10*th.*—Morning, P. 88, resp. 20, temp. 99°. Evening, P. 100, resp. 22, temp. 101°.

October 11*th.*—Morning, P. 104, resp. 21, temp. 99°. Evening, P. 104, resp. 25, temp. 102$\frac{2}{5}$°.

October 12*th.*—Morning, P. 92, resp. 21, temp. 99°. Evening, P. 108, resp. 24, temp. 101⅖°.

October 13*th.*—Morning, P. 92, resp. 17, temp. 99°. Evening, P. 104, resp. 26, temp. 102°.

October 14*th.*—Morning, P. 104, resp. 19, temp. 99⅗°. Evening, P. 104, resp. 22, temp. 101°.

October 15*th.*—Morning, P. 104, resp. 19, temp. 98⅖°. Evening, P. 116, resp. 23, temp. 99°.

Remarks.—In the first place, in regard to the intestinal ulcer, the history of malaria and the absence of all evidence of syphilis or tuberculosis lead us to conclude that the ulcer is dysenteric. This case will swell the number of cases of latent intestinal ulceration; but yet it is probable that an investigation in this direction might have elicited from the patient a history of dysentery in the earlier periods of the disease.

It is surprising to note the slight constitutional disturbance that this extensive suppuration gave rise to. The most reasonable diagnosis seemed to be low continued malarial fever, with congestion of the liver. When I was told of the hemorrhage and death, I changed the diagnosis to typhoid fever of the variety called *ambulatorius*. Here was the error. At this time the diagnosis of abscess of the liver might have been made, because there were no grounds for a diagnosis of typhoid fever in the face of such a temperature-sheet, and of the history of the case. The patient complained so little of hepatic pain that the symptom had been allowed to fall into the background.

Probably the diagnosis would have been of little service, since I do not believe that the aspirator could have been successfully used.

It is of interest to note the absence of an embolus in the portal veins. I take it that the embolus in these cases belongs to the infecting variety, and breaks down into pus, together with the surrounding tissue, contrasting with the mechanical embolus of the pulmonary artery, that I exhibited some time ago, where the plug remained intact, and the wedge-shaped area of pulmonary tissue beyond was breaking down by a process of gangrene.

Dr. G., in reply to the question as to what were the symptoms in abscess of the liver, said that the disease was very apt to be latent. In two cases of abscess of the liver that he had seen previous to this one, the lesion had not been detected during life. In one a small abscess had opened into the pleural cavity, and the resulting empyema only was diagnosed. In the other, the abscess was enormous; it had for the upper wall the deeply-excavated left lobe of the liver, and the inferior wall was made up

by the adherent stomach. The dyspeptic symptoms in this case were so slight, that the diagnosis of cancer of the stomach, determined upon by one of our best diagnosticians, was considered very doubtful.

In the present case, several symptoms were absent or imperfectly developed. The man was living in the temperate zone. The fever was not wearing him out, as is the case in suppuration; in fact, but little attention would have been paid to it had it not been for the thermometer. There were no localized bulgings, no fluctuation. The pain in hepatic abscess may be much better marked than in this case,—extending up to the right shoulder and accompanied with marked tenderness when the inflammatory process involves the peritoneum. Symptoms of obstruction of the duct or of the portal vein may or may not exist. The failures of diagnosis probably explain why many of these symptoms are not elicited from a large number of the cases.

Dr. Henry asked if there was any enlargement of the mesenteric glands. In a case of peritoneal tuberculosis reported by him to the Society three or four years ago, the peritoneum covering the intestines was studded for many feet with miliary tubercles. The mesenteric glands at the spinal attachment of the mesentery were matted into a tumor several inches in circumference. There was almost incessant colliquative diarrhœa, though the intestinal mucous membrane appeared healthy throughout. In another case, the intestine was the seat of several tubercular ulcers, the mesenteric glands were *not* enlarged, and there was no diarrhœa. He had explained the diarrhœa in the first case by a paralytic pressure upon the sympathetic nerves supplying the intestine, and in support of this view referred to the well-known experiments of Moreau. The present case furnished still further negative evidence in support of this view.

October 25th, 1877.

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3. *Chronic hepatitis with enlargement.*

Presented by Dr. John Guitéras.

George W., æt. 43, German, farmer, resident of Manayunk, contracted a diarrhœa during the war, which has always shown some tendency to return. Has had malaria; very probably has had syphilis; otherwise has been a very healthy man. No family history of cancer or phthisis. Admitted to the Philadelphia Hospital on September 2d, 1877, with well-marked jaundice. He was somewhat emaciated; the skin was very yellow, showing about the face and neck a good deal of redness. He also pre-

sented the symptoms of itching, dark-colored urine, feeling of languor and sleepiness, and a small, slow, and feeble pulse. He was up and about.

His skin has been of the same color, with slight changes, since the beginning of February of this year. He never had jaundice before, and gives no history of biliary colic. The jaundice came on gradually, preceded by several days of diarrhœa, but apparently no gastro-duodenal symptoms. Since the existence of the jaundice his abdominal symptoms may be summed up as follows: nausea, sometimes with vomiting; flatulence, pyrosis, and, most constant of all, a sense of fulness about the epigastrium. These symptoms were relieved at intervals by diarrhœa setting in. The stools have been at times clay-colored, at times normal. Dr. Musser's notes were carefully taken throughout the patient's illness. They show no change in the condition of the patient except slight changes of color, and aggravation or amelioration of gastro-intestinal symptoms. The diarrhœa became more constant. Some swelling of the feet was noticed at times. At times also he complained of dimness and yellow vision. The abdomen was generally distended, but no ascites could be detected. During the month of December his treatment consisted of iodide of potassium and bichloride of mercury, alternating with ipecac and bicarbonate of sodium.

October 2*d*.—There had been no improvement. He was ordered muriate of ammonia gr. xv every three hours, and cloths saturated in a solution of the same were applied over the liver. At this time the area of deep hepatic dulness extended from the fourth interspace to two inches below the margin of the thorax in the line of the nipple. There was no pain or tenderness. The margin of the liver felt smooth and resisting.

This treatment gave some relief; it certainly cleared up the skin considerably, and reduced slightly the size of the organ.

On the 10th the jaundice returned, and he was ordered jaborandi. He got an overdose of the drug, and its action was followed by much prostration. Yet there was no tendency to progressive wasting and prostration until the 15th, when he began to suffer with uncontrollable hiccough. The treatment was changed to nitromuriatic acid, strychnia, and pepsin. From the 15th to the 25th, every form of treatment was used to relieve the hiccough. The interrupted current, applied to the neck and diaphragmatic region, gave some relief. Finally the oil of cajeput seemed to arrest the hiccough. The hiccough, the prostration, and the temperature made the diagnosis of diaphragmatic pleurisy a question of great interest.

On the 27th I noted slight pleural friction-sounds at the right base. On the 28th the proper significance was given to these sounds. My note

of that day reads as follows: No pain or nausea; slight diarrhœa; pulse weak and small; tongue red and angry looking; impaired resonance at right base posteriorly, commencing at the angle of the scapula; flatness extends from tenth interspace to the margin of the ribs; there is no alteration in the shape or movements of the chest; inspiratory sound at the right base posteriorly is slightly interrupted, feeble, and terminates with fine crackling râles. The same râles are heard at the left base, and at both places almost disappear after deep respirations.

He emaciated rapidly; the diarrhœa became uncontrollable and accompanied with much tenesmus, aggravated by hemorrhoids. These symptoms were relieved by injections of nitrate of silver into the bowel. He fell into a semi-typhoid state, with slight wandering delirium, from which he could always be aroused, and finally died of exhaustion, November 4th.

The urine contained no albumen or sugar. It was normal in amount, except when the diarrhœa was excessive. It always was high-colored, and responded to the tests for biliary salts and pigment. Leucine and tyrosine were found after slightly concentrating the urine.

Post-mortem.—The skin is decidedly yellow, as are also the internal organs. There is a slight effusion, also stained, in the peritoneal cavity. The liver weighs five pounds three ounces. Its peritoneal covering is smooth and shining, except in some places about the edges, where there are small patches of opacity, evidently due to distentions of the organ. The liver overlaps the stomach considerably, and extends to within one and a half inches of the umbilicus. The shape of the organ is not changed. The organ presents a deep-green color. The acini are not well defined; the central hepatic veins appear darker than the surrounding tissue, but the contrast is by no means distinct. The tissue of the liver is very firm, presenting fine cirrhotic contraction in some spots about the anterior edge. The section is, nevertheless, perfectly smooth. Iodine does not reveal any amyloid degeneration. The gall-bladder contains a small amount of inspissated black bile. No obstruction can be found, except at the duodenal end, in the biliary passages, though dissected some distance into the liver-substance. The swelling of the duodenal mucous membrane and some mucus present might have given rise to obstruction; but there was no dilatation of the tubes, and they were empty. There are no syphilitic cicatrices. Under the capsule one very small piece of calcareous matter was found, not imbedded in cicatricial tissue. The mucous membrane of the duodeum is congested, thickened, and covered with an abundant mucous secretion. The mucous membrane of the remaining portion of the small intestine is also congested. The large

intestine shows considerable thickening of the mucous membrane, is grayish in hue, and of a uniformly fungoid velvety appearance. The kidneys are healthy; the spleen enlarged and congested. All the tissues are stained with bile. The heart is healthy, save slight thickening of the aortic semilunar valves. The lungs are healthy, with the exception of hypostatic congestion of the lower lobes posteriorly. The pleuræ are not adherent.

Remarks.—One of the most interesting features of the case is the temperature. It was noted from October 21st to the time of death, as follows:

Oct. 21.—M., Temp. 100°, P. 92, R. 17. E., Temp. 98°, P. 76, R. 17.
Oct. 22.—M., Temp. 98°, P. 80. E., Temp. 103°, P. 92.
Oct. 23.—M., Temp. 95 2–5°, P. 80, R. 20. E., Temp. 100°, P. 76.
Oct. 24.—M., Temp. 101 2–5°, P. 112, R. 23. E., Temp. 96°, P. 72.
Oct. 25.—M., Temp. 93°, P. 86, R. 17. E., Temp. 101°, P. 96.
Oct. 26.—M., Temp. 95°, P. 76, R. 19. E., Temp. 94 3–5°, P. 21, R. 18.
Oct. 27.—M., Temp. 103°, P. 116, R. 30. E., Temp. 98°, P. 92, R. 18.
Oct. 28.—M., Temp. 95 1–5°, P. 84, R. 15. E., Temp. 100°, P. 96, R. 18.
Oct. 29.—M., Temp. 97°, P. 92, R. 15. E., Temp. 98 2–5°, P. 96, R. 14.
Oct. 30.—M., Temp. 97°, P. 84, R. 14. E., Temp. 98°, P. 88, R. 16.
Oct. 31.—M., Temp. 94 2–5°, P. 72, R. 14. E., Temp. 96°, P. 68, R. 17.
Nov. 1.—M., Temp. 99°, P. 92, R. 21. E., Temp. 93°, P. 72, R. 18.
Nov. 2.—M., Temp. 95°, P. 92, R. 21. E., Temp. 96 3–5°, P. 96, R. 22.
Nov. 3.—M., Temp. 91 2–5°, P. 76, R. 11. E., Temp. 91°.

With such a temperature, and with a rapid emaciation, the exclusion of suppuration became a question of interest, the more so since the hiccough and physical signs at right base pointed to diaphragmatic pleurisy,—a form of pleurisy that terminates not unfrequently in suppuration. But the temperature sheet shows a regularity that is not characteristic of suppurative fevers. I find that every third temperature noted is pretty regularly a high one, the fall being very great in the two intervening temperatures; so that the rise and fall do not present the usual relations to the morning and evening hours. The very chronic nature of the trouble, the absence of a cause, together with the above temperature, the persistent jaundice, and passive nature of the enlargement, excluded abscess. I excluded diaphragmatic pleurisy because there was no dyspnœa, pain, or anxiety, and because the localized spots of tenderness near the xiphoid cartilage, and at the root of the neck along the course of the phrenic nerve, were absent. The râles heard at base of right lung were thought to be due to want of expansion of the lung from pressure. The hiccough might have been due to the same cause, or to central disturbance from toxæmia.

The curious range of temperature may be due to an intermittent absorption of effete products from the liver, or an intermittent arrest of the oxygenating processes going on in the liver, an arrest that must influence the general temperature if we remember that in health the temperature of the organ reaches 106°. It is to be regretted that the amount of urea excreted was not observed.

I may observe that I do not believe the electric current passed along the phrenic nerve. I noticed that the diaphragm continued its normal movements, and only the muscles of the abdominal and thoracic walls were seen to contract under the influence of electricity. I believe that the shock produced by the current gave the temporary relief, and suspect that a strong current applied to the thighs would have had the same effect.

The diagnosis during life was chronic catarrhal jaundice resulting in hyperplasia of the connective tissue. It is very rare to find such marked jaundice in the early stage of cirrhosis, or stage of enlargement, and it is probable that the catarrh of the tubes was the primary lesion. I thought during life, and think so yet, that this liver might be likened to the kidney in the second stage of Bright's disease, so far as the anatomical differences between the two glands will allow.

The jaundice and absence of involvement of other organs excluded the amyloid liver; and the passive, smooth enlargement excluded cancer. The empty and undilated tubes, the almost empty-gall bladder, show that there was no obstruction at the duodenal orifice or in the larger tubes.

Dr. Tyson said this was the first time he had ever known human urine to respond to Pettenkofer's test for bile as ordinarily directed to be applied. From the directions usually given for using this test in the books, we are led to believe that the elements need but to be added to urine containing bile to have a prompt reaction. But this is not the case. Some years ago, in making a number of experiments with this test he was only able to obtain the characteristic reactions by adding ox-bile to human urine. The test is for biliary acids, and these should first be extracted, redissolved, and tested for. Dr. T. recalled, however, a case of strangulated hernia, which was operated upon, and after recovery from the operation it was thought desirable to ascertain if the fecal matter came from the upper part of the small intestine. It was thought that if it did it would contain bile. Pettenkofer's test was employed, and responded promptly and satisfactorily.

Dr. Guitéras replied that he himself was very much surprised at the result obtained in this case by the employment of Pettenkofer's test, since he had never previously, in using this test, obtained satisfactory results.

There was, however, no doubt as to the complete and convincing reaction of the test in the present case; the purple band between the acid and urine and sugar was quite distinct.

Dr. Longstreth said he wished to call attention to the form of cirrhosis occurring from a malarial cause; in such cases the indurative form prevailed, and the organ was usually not much reduced in size, but simply firmer and harder than normal. The color of the specimen before the Society was much darker than that found in livers of patients jaundiced from a catarrhal inflammation of the bile-ducts. This increased intensity of coloration would point to the presence in the liver-tissue as well as the blood of a pigment such as is found in chronic malarial poisoning. It could, of course, be accounted for by the presence of bile-pigment solely. He has a specimen in the museum of the Pennsylvania Hospital removed from a patient suffering from chronic malarial poisoning, with jaundice, which presents conditions similar to the present specimen. In the specimen referred to, the three forms or degrees of cirrhosis of the liver were present, and were well illustrated by the organ, viz., simple induration, lobulated contraction, and also a few hob-nail masses. The liver was slate-colored, and there were seen also numerous black points; the spleen was diseased, and the blood contained pigment-matter.

The pathology of cirrhosis of the liver from simple catarrhal inflammation of the bile-ducts is a doubtful matter. May not the cirrhosis of the organ presented to-night be due to malarial poisoning?

Dr. Tyson thought this liver not unlike a pigmented malarial organ, and that pigment-particles would probably be found in the cells if examined microscopically.

Dr. Guitéras said that he had excluded malaria because there was no history of malarial cachexia, and the patient had had no malaria since the war. The spleen was perfectly healthy, and he did not think that the deep-green color of the organ, somewhat changed since the *autopsy*, pointed in that direction. He suspected the microscope might not be of use in determining this question, because in cases of intense jaundice, granules of biliary coloring-matter were found in the liver-cells. He thought the slate color was not present in the specimen. Dr. G., in answer to Dr. Allen, who asked if any of the members had seen enlargement of the veins during cirrhosis of the liver, and especially those surrounding the umbilicus, replied that he attached much importance to this symptom, viz., enlargement of the veins, as an evidence of portal obstruction, especially in cirrhosis, where he had almost always found it in advanced cases. The enlargement of the veins around the umbilicus he had never noticed,

though early writers speak of them as forming a distinct areola. Dr. G. replied to Dr. O'Hara that he did not feel justified in ascribing the relief of the hiccough to the use of oil of cajeput. Ten drops were given every two hours. He expected the action of this drug to be rapid, and yet the hiccough did not stop until eight or ten doses were given.

November 8th, 1877.

Report of the Committee on Morbid Growths:

"The liver presented by Dr. Guitéras, upon microscopic examination, shows the cells, situated mainly at the periphery of many of the lobules, to be colored greenish yellow, increased in size, and slightly more granular than normal; also at the periphery of a few lobules the cells are seen to be enlarged and infiltrated with oil-globules. There are not seen any pigment granules, such as are found in melanotic disease due to malarial poisoning. The fibrillar connective tissue, especially that found in the interlobular spaces, is decidedly increased, and infiltrated with numerous embryonic cells. The pathological change is one of indurated hepatitis of the first stage, or passing into the second, with a staining of the cells by biliary coloring-matter." *November 22d*, 1877.

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4. *Cirrhosis of the liver.*

Presented by Dr. F. H. Gross.

On the 22d of last month (October) I was requested by a medical friend to see a patient, with the object of performing the operation of paracentesis abdominis; but, as the gentleman alluded to was himself ill, I did not have the opportunity of meeting him at the bedside of the patient. The latter I found to be a German, aged 42 years, and by occupation a dealer in shoe-findings. He was greatly swollen from effusion into the peritoneal cavity, and from anasarca of the lower limbs and scrotum. There existed also a reducible umbilical hernia of the size of a large fist. The urine was high-colored, not scanty, but deposited a copious sediment of lithates. He had felt ill at irregular periods for a year or so, and during that time occasionally consulted a physician, but was not confined to his room and bed until about five weeks prior to my first visit. During the earlier stages of his disease he is said sometimes to have had an icteroid appearance, and about a month before my seeing him a marked attack of jaundice, but this had entirely disappeared. So far as I could learn, the real nature and seat of the disease had not as yet been made out.

The gentleman at whose request I saw the patient, and who, on account of his own temporary illness, felt induced to relinquish the case, had been in attendance only a short time.

In regard to the patient's habits I learned that he drank beer regularly, and occasionally light wines, and at times, when suffering from depression of spirits, which is said not to have been very frequently, he would indulge more freely in beer, but was never addicted to the use of the stronger alcoholic liquors. Though not markedly emaciated, he was now very weak, mentally as well as physically. His memory being unreliable, he was able to give only a vague account of the disease, or of the train of symptoms that had manifested themselves. I regret, therefore, that the history of the case is necessarily incomplete. We may assume, however, that it did not differ materially from the generality of cases of cirrhosis of the liver uncomplicated with organic disease of other vital organs. He had often complained of a general sense of discomfort and of dull pains in various parts of his abdomen, and during the past summer felt that this part of his body was enlarging. At that time there was already some dyspnœa, especially on making any bodily exertion. He was then told that he had the asthma, and later on that he was getting the dropsy. Whatever doubt there may have been concerning the former, the latter was certainly true, as I drew off at my first visit, by the operation of tapping, nine quarts of pale-yellow and slightly turbid serum.

In regard to the diagnosis, I have to say that at my first examination of the patient, and more especially after the drawing off of the peritoneal effusion, when a more satisfactory physical exploration of the abdominal as well as the thoracic viscera could be made, and from the hardened feel of the liver below the edge of the ribs, I suspected no other disease than cirrhosis of that organ. And when some days later an examination of the urine showed no trace of albumen, and a renewed exploration of the organs of the chest revealed no organic disease there, I felt strengthened, by a process of exclusion, as it were, in my original inference, and the existence of cirrhosis of the liver was as clearly established as could be without positive demonstration.

The prognosis, as a matter of course, was unfavorable, and the treatment only palliative. Upon the administration of diuretics and benzoic acid with borax, the secretion of urine became more copious, of lighter color, and ceased on cooling to show any sedimentary deposits. Such tonics, and also as generous a diet as the stomach would tolerate, were allowed. From the opening made by the trocar, fluid continued to ooze for about eight days, which was absorbed by the application of sponges

and cloths. The swelling of the abdomen was, therefore, kept down, and the anasarca of the lower extremities and scrotum disappeared. The debility of the patient was, however, steadily progressive, and he finally died of exhaustion on the 8th of November.

Autopsy, about thirty-six hours after death.—On opening the abdominal cavity the liver was noticed at once to be the seat of disease. It was hardened, and had a firmer feel than in the normal state. Its color was of various tints of yellow, and it presented upon the greater part of its surface numerous rounded elevations of different sizes, giving the so-called hob-nailed appearance. The corresponding depressions indicated the existence of fibrous tissue, compressing the lobular structure. Its weight was four and a quarter pounds. The gall-bladder contained a quantity of dark bile of a thick consistence, which imparted a sandy feel between the thumb and fingers. The kidneys were of normal size, and appeared to be entirely healthy in their structure, as did the other organs of this cavity. The thoracic organs were healthy. To Dr. J. Collins, who assisted at the *post-mortem* examination, I predicted the finding of a cirrhotic liver.

The following is a note from Dr. Seiler of the microscopic examination:

"A section of the liver made with a Valentine knife, and stained with carmine, shows large bands of connective tissue surrounding the lobules, while the liver-cells were mostly in a state of advanced fatty degeneration, many of them having been supplanted by fat-globules."

Dr. Seiler said he wished to show, in connection with cirrhosis of the liver, a microscopic section made from the liver of a cat, in which could be seen the physiological changes occurring in this disease. There is a very decided increase of the connective tissue, forming wide bands between the lobules of the organ; the liver-cells, however, are found to be perfectly healthy; there is not any fatty degeneration observable. Dr. S. thought this was a demonstration of an inflammatory condition of the liver causing cirrhosis, without any fatty degeneration; also that we may have this pathological change in the organ without intemperance or syphilis being the exciting cause. *November 22d*, 1878.

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5. *Perforating typhoid ulcer of ileum.*

Presented by Dr. F. P. Henry. Clinical history by Dr. Louis Starr.

C. G., æt. 31, an Alsatian, a cabinet-maker by trade, was admitted to the male medical ward of the Episcopal Hospital on November 2d, 1877. His health had been good until two weeks before admission, when he began

to suffer from headache and general malaise; there was also a tendency to diarrhœa, and upon several occasions he had slight hemorrhages from the nose. On October 31st, he became too sick to work, and upon coming to the hospital, two days later, he presented the ordinary features of a case of typhoid fever of moderate severity. During the first five days of treatment, complaints were made of abdominal pain, and the diarrhœa was more than usually profuse; at the end of this time, however, both symptoms abated, the stools being reduced to one or two in twenty-four hours, and the disease appeared to be progressing very favorably.

At noon on November 11th, the tenth day of treatment and the twelfth day of the attack, the patient was suddenly seized with severe pain in the right iliac region; one hour later, he had a severe chill, accompanied by the discharge of about half a pint of dark, partially clotted blood from the bowel. Throughout the succeeding twenty-four hours there were eight hemorrhages from the bowel, the quantity of blood lost being estimated at nearly six pints. There was *slight* distributed tenderness of the abdomen, the respiration was chiefly thoracic, and deep inspiration caused pain at the epigastrium, but there was little or no abdominal distention. The general symptoms were those of collapse. The intellect remained clear almost up to the moment of death, which occurred at 2.30 P.M. on November 12th.

I am indebted to Dr. Starr for the preceding clinical history, and for the opportunity of presenting this specimen.

I have already read two papers before this Society on the intestinal lesions of typhoid fever, with special reference to the occurrence of perforation, in which I maintained that peristalsis is abnormally excited in typhoid fever, and that this abnormal peristalsis occurring at a critical period of the disease, viz., when the patches are at their acme of medullary infiltration, might convert what would otherwise be a gradual molecular ulceration, into a sloughing *en masse*, and thus favor perforation. I also maintained, and do still, that peristalsis favors perforation not only in the manner just described, but also in all three of the modes in which, according to Murchison, this accident occurs,—viz., "1, by rupture of the attenuated coats; 2, by a continuance of the ulcerative action leading to a pin-hole perforation; 3, by sloughing of the entire thickness of the bowel, and the dropping out of the sloughs." The gradation of the lesions I explained by the distribution of the superior mesenteric vein. It was argued, in opposition, that a perforation frequently occurs high up, and in states of constipation; but for fuller details I refer to the last volume of our Transactions.

Since my last paper I have analyzed twenty cases of perforation with reference to its association with diarrhœa, its situation, date of its occurrence, and the time elapsing between the first symptoms of the accident and the time of death. The cases are not numerous, but are interesting, because they are, I believe, the only ones that have been analyzed from these points of view, and because they afford a very decided confirmation of what I advanced on more strictly theoretical grounds.

Sixteen of the cases are reported in the first twenty-seven volumes of the Transactions of the Pathological Society of London, and three in the Transactions of the Pathological Society of Philadelphia. The case just reported makes the twentieth. In five of the cases there is no statement with reference to the condition of the bowels; in one the bowels were "regular;" in thirteen there was, either throughout or at some period of the disease, diarrhœa of all grades of intensity, from "profuse" and "urgent" to "slight."

In one of these cases perforation occurred during a relapse; in the primary attack there was diarrhœa, none during the relapse. In another there was at first constipation, for which compound jalap powder and castor oil were given! It is scarcely necessary to add that diarrhœa set in, nor is it at all to be wondered at that perforation occurred. In one case only was there a generally constipated state of the bowels. This is one of Dr. Murchison's cases, and is to be found in vol. xvii. of the Transactions of the Pathological Society of London. This, therefore, is the only case I have yet encountered that seems to militate against the peristaltic theory; and even that can be fully explained. The patient was a male, æt. 14, and death occurred "on the forty-second day after apparent convalescence." He was gaining strength when, on the thirty-ninth day, he "had pain in the stomach relieved by pressure." . . . "It is worthy of note that the bowels had been constipated for several days." The author evidently considers the thirty-ninth day as the date of perforation, when the patient "had pain in the stomach relieved by pressure." Probably the pain was of a colicky nature, due to peristaltic efforts to impel the solid fæces along the intestine, and this *peristalsis* led to perforation. After perforation the pain is not "relieved by pressure;" and it is worthy of especial note that "on the following day the pulse rose to 124, the belly *became* very distended and tender," etc.

In the twenty cases there are twenty-seven perforations. In the present case, it will be noticed, there are three, very near together. In five cases the perforation was situated in the large intestine, as follows: in one case the vermiform appendix was perforated; in a second there was one per

foration in the colon three and a half inches below the valve, and two in the sigmoid flexure; in two other cases there were single perforations of the sigmoid flexure; and in the fifth there was a perforation of the cæcum, communicating through an abscess with one in the ileum. As regards the seat of perforation in the ileum, in one case this is not stated; in another it is vaguely described as being in the "lower part" of the ileum. In the cases in which it is accurately given it varied between four feet above the valve and two inches above it, as in the present case. Eight times it occurred at twelve inches from the valve, or within that distance. In the perforation four feet above the valve the accident occurred in a relapse, and on the fortieth day from the beginning of the first attack. The perforation, although absolutely high up, was not so relatively, for it occurred within a foot of the primary cicatrized ulcers.

The insiduous manner in which perforation often occurs is shown by the fact that in five of the twenty cases the date of the accident was not determined, owing to the absence of explosive symptoms. In the remaining fifteen cases it occurred at different periods,—the earliest being on the seventh day, the latest in the seventh week,—and this irregularity affords a strong proof that some extraneous circumstance, something not essential to the disease, is the cause of this accident. This something I believe to be peristalsis, principally.

The frequency of hemorrhage in cases of perforation is very striking, this hemorrhage occurring before, at the time of, or after the accident. I cannot give the exact ratio of cases of perforation in which hemorrhage occurs, as I have not tabulated it, but it is large. Taken in connection with the cases in which hemorrhage occurs without perforation, this complication is to be regarded as a premonitory symptom of that accident, and prophylactic treatment accordingly instituted.

The hemorrhages occurring in connection with perforation are generally profuse, as no large arterial twig is eroded until the ulcer has nearly perforated. In speaking of hæmatemesis as a symptom of gastric ulcer,—and the statement is here almost equally applicable,—Leube says, "The most copious hemorrhages naturally occur from erosion of a large vessel, a symptom which can occur only when the ulcer, not necessarily a large one, has perforated all the tissues of the stomach." I would amend this statement by the addition of the word "almost," so that it would read, "when the ulcer has perforated almost all the tissues," for copious hemorrhage from the bowel in typhoid fever is frequently recovered from, and perforation almost never.

The time elapsing between the first symptoms of perforation and death

could be determined in fourteen cases, and varied between a few hours and fifteen days, as follows: in four cases, within twenty-four hours; in five, within forty-eight hours; and in the remainder, three, four, five, seven, and fifteen days after.

Dr. Roberts said there had been no reference to perforation of the intestines occurring in walking cases of typhoid fever. He was able to recall the case of a man who presented himself at the Pennsylvania Hospital complaining of abdominal pain; he had been sick about twelve days, and, having no passage from the bowels for about a week, had the night previous to coming in the hospital taken a purgative. After being admitted into the hospital, more marked symptoms of peritonitis set in. Dr. Meigs, who was on duty at the time, thought the case would prove to be one of walking typhoid fever. The patient died in six days, and the *autopsy* showed perforation of the intestine, about six inches above the ileo-cæcal valve, with general peritonitis, which was thought at the time to be due to the purgative.

Dr. Henry replied that he had, two or three years previously, reported a case of walking typhoid fever, which would be found in the Transactions. The man, a patient in the Episcopal Hospital, during his illness continued daily to walk about the wards. His only complaint was of vertigo. Typhoid fever was suspected, and he was kept on liquid diet. Death occurred from perforation of the ileum. *February 22d*, 1878.

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6. *Cancer of the œsophagus.*

Presented by Dr. John Guitéras.

Though I did not have an opportunity to study carefully during life the patient from whom the specimen was removed, yet there are some points about it that may be of interest. She was an old woman, presenting considerable cachexia. She had had difficulty in swallowing for several months. There was no history of cancer in the family. I saw her once in the wards of my colleague Dr. Wood. She vomited all solid food, and there was constant gurgling in the larynx. She was so frail, and was so much disturbed by the attempt to pass an œsophageal bougie, that the resident physician was obliged to give it up. There was some obstruction in the air-passages. Auscultation proved it to be mainly seated in the left bronchus. The cachexia, the persistent dysphagia, which was primary to and much more marked than the dyspnœa, and the absence of signs of

atheroma and aneurism, were the symptoms pointing to cancer of the œsophagus. Besides, my attention has been called by Dr. Allen to the frequency with which cancer develops in the œsophagus opposite the bifurcation of the trachea. In the complete cartilaginous ring at this point he finds a source of irritation to the tissues of the œsophagus. The slight inclination of the œsophagus to the left explained the greater obstruction in the left bronchus. The specimen now before the Society shows very well how this pressure was brought about. Besides this anatomical relation, it will be seen that the tumor has attained its greatest size in the left wall of the œsophagus. *December* 13*th*, 1877.

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7. *Excision of four inches of the upper part of the rectum in consequence of intussusception caused by a villous tumor.*

Presented by Dr. M. O'Hara.

I was called, October 23d, 1877, to Catherine Williams, 72 years of age, a native of Ireland, suffering from a hemorrhage from the bowels. She stated that, while defecating, a large mass was extruded suddenly from the anus, which bled very much, and which she could not replace; that three months previously it had appeared in the same manner with bleeding, but she was able to replace it; since that time she was subject to costiveness (easily relieved by the use of pulv. glycyrrhizæ comp.): she had losses of blood with her stools, but no protrusion since that time.

She had a cancerous family history,—a sister having died of cancer of the breast, and a brother having been affected with epithelial cancer of the nose.

On examination there was found a fungous, cauliflower-like mass hanging outside the anus, very red, with an apparent pedicle extending within the rectum, six inches long, and to the feel freely supplied with blood-vessels. On consultation with Dr. Willard, the growth was pronounced malignant and its removal demanded. The tumor was three inches long, two and a half inches wide, and one inch in thickness, and was supposed to project from the anterior wall of the rectum, high up; the apparent pedicle was considered to be a portion of this wall dragged down by the weight of the tumor through the other portions of the rectum. Great difficulty was experienced in the diagnosis, as the tumor was large and obstructed the orifice of what was afterwards ascertained to be an intussuscepted portion of intestine, and not the ordinary pedicle of a tumor. The mass

was soft, friable, bled freely, and had somewhat the feel of placental tissue. The lower part of the rectum seemed to be in a healthy condition as far as the finger could reach. With the finger you appeared to touch the point of attachment of the apparent pedicle on the anterior wall; behind, the attachment was not so distinctly felt.

The patient had no appearance of cachexy, or any signs other than of good health.

On the 24th of October, the tumor and portion of bowel (about four inches) presented to your notice were removed by the écraseur, and the cut edges of the intestine were brought down and tacked with sutures to the verge of the anus. There was no hemorrhage during or after the operation. The patient was given opium freely by mouth and rectum. During the first twelve hours she took as much as twelve grains, and afterwards a grain every three or four hours as needed to cause sleep and suspend the action of the bowels. Fluid nourishment was given freely. There was no sign of peritonitis, or even much febrile irritation, during the whole of the after-treatment.

October 26*th.*—Flatus was passed freely, showing a complete thoroughfare. Pulse 100. Wakes up and takes nourishment.

27*th.*—Comfortable; takes nourishment; passes wind freely. The stitches were removed, and the gut permitted to ascend, which it did to about two inches.

29*th.*—Had a small, consistent, well-formed fecal evacuation; is doing well; pulse 82; slightly reduced in strength; some abdominal distention from flatus. Takes quinine, iron, etc., and opium is kept up regularly.

November 1*st and* 2*d.*—Doing well; daily normal evacuations.

6*th.*—Doing well; bowels regularly moved. Has appetite, and takes food regularly.

12*th.*—Ceased attendance. Upon examination this day the rectum was found capacious; the mucous membrane apparently soft and free from disease; no nodules or ulcerations; as high as the finger can reach, and with difficulty, was felt the orifice of the divided intestine, somewhat indurated, and surrounded with a ring of inflammatory deposit. The first finger could be carried with some difficulty into the opening.

December 11*th.*—The patient feels as well as before the operation; is going about her household duties as before; sometimes a little costive and takes castor oil, but her bowels are generally moved naturally.

This case seems unique. I find in authorities no details of a similar case. Aitken, "Science and Art of Medicine," in his article on intussusception states that cases of intussusception in adults are rare,—so rare,

that in the extensive experience of one of the largest civil hospitals in London (Guy's) Dr. Wilkes records that he has never seen but one case; and in this case the obstruction was never complete, and death did not occur for some weeks. In the Transactions of the Pathological Society of London for the first fifteen years of its existence, only seven cases are reported, no two occurring in the individual experience of any one man. In one case the symptoms continued for three months, ending in the passage of an invaginated portion of the ileum (containing a polypoid tumor) by the rectum, with recovery. Aitken says he never saw a case during life, or a *post-mortem* of one, in an adult. Five cases are reported in the Transactions of the Pathological Society of London, which were associated with polypoid tumors at or near the site of lesion. He gives twelve per cent. as the proportion of the colic invaginations. In this connection I would refer to a case of invagination of the descending colon, with *autopsy*, reported by Dr. G. W. Brown, of Port Carbon, page 729 of Transactions of the Pennsylvania State Medical Society for 1877. In this case, during life, the trouble was diagnosed scirrhus of the bowel. The symptoms of obstruction existed off and on for one year. The *autopsy* showed an invagination six inches long, caused by a tumor two inches long. The fæces had caught the tumor, drawing the bowel into itself. The inverted end of the intestine resembled the os uteri, with the large cap of the tumor outside, and the body entirely stopping up the opening, which was the immediate cause of the trouble. There had been frequent attacks of inflammation, fibrous bands had formed, and bound the parts together; this, with obstruction of the tumor to the passage of fæces, caused perforation, escape of the fæces into the peritoneum, peritonitis, and death. In my case similar results must have happened if the trouble had not occurred in a part within reach of surgical art. Similar cases should be thought of in the treatment of invagination by injections or forced insufflation (with bellows), which would only make the obstructing cause act more injuriously, increasing its stop-valve action. I find no record of a similar case to the one here reported, of recovery after surgical operation, though higher up in the bowel nature has sloughed off the invagination and tumor, with recovery.

I am indebted to Dr. Seiler for the following examination of the growth: "The piece of rectum sent to me presents, on section under the microscope, the appearance of hypertrophied villi, lined with columnar epithelium, and penetrating into the submucous tissue. The medullary substance of these villi appears to consist of only a few fibres of connective tissue."

Dr. Willard said, When this patient was first seen, the anus was found

to be easily dilatable, and with the index and middle fingers the attachment of the mass to the anterior wall of the rectum could be readily felt, some four or five inches above the anus, this attachment being within reach on account of the relaxed condition of the tissues of the perineum. Posteriorly the connection with the bowel was not so distinct, a fact which is now easily explained, but at the time this circumstance, taken in connection with other appearances, caused me to believe that the tumor had its origin from the anterior wall.

The soft, fungous condition of the mass, its tendency to bleed, and the family history of the case, indicated that it was malignant in its character, and removal seemed to be the proper course to be pursued. Accordingly, the chain of an écraseur was passed around the apparent pedicle, and was caused to cut its way slowly through the tissues. No hemorrhage followed, but when the removed piece was examined it presented the puzzling appearance of being a tube four inches long, of the size of the large bowel, but, instead of the normal condition of being lined by mucous membrane, its inner wall was found to be serous, while the outside was mucous membrane. This is difficult of explanation except upon a model; and I have here a muslin tube which will represent the intestine, the different-colored sides indicating respectively serous and mucous membranes. When I invaginate a portion of the tube, and then cut it across, it will be seen that, counting the wall to consist of only two layers, serous and mucous, first is encountered a serous membrane, then a mucous membrane, then the same again, then peritoneum, then the same again, then mucous membrane, and the centre of the gut is reached. The other side will be the same. When cut across and removed, the incised peritoneum seems to present the appearance of a ring or cul-de-sac, but upon relieving the intussusception the appearance presented is the exact counterpart of the specimen under consideration. If any one of you will try this manœuvre, you will see that it perfectly answers the requirements of the case.

Recognizing the fact that a complete excision of about four inches of the upper portion of the rectum had been made, two silk threads were passed through the upper portion, and the cut edges brought down and secured loosely to the verge of the anus, the intention being to hold the bowel in position until inflammatory adhesions should have glued it in a safe position. Here it was kept until the third day, when it was permitted to ascend about two or three inches, the threads acting as reliable guides and as preventives against too great recession. The anus was not freshened, as to have retained the cut end at this point permanently would have caused too great tension upon the bowel.

The happy result obtained was doubtless due to the condition of a small tube entering into and far underlapping a larger one, just as is sometimes seen in tin water-pipes. Had the rectum been allowed to become full during the first few days, or had diarrhœa supervened, overflow into the peritoneal cavity would doubtless have occurred. The opium, however, controlled everything nicely, and not a single bad symptom occurred during the progress of the case.

Dr. John Ashhurst, Jr., asked at what distance from the anus the growth was supposed to have originated, and at what height the reflexion of bowel was found. From the history, as read, and from his inspection of the specimen, he was somewhat at a loss to know why the case was called one of intussusception, rather than one of prolapsus of the rectum, which, as was well known, might be produced by the straining efforts excited by the presence of a polypus or other tumor in that part of the bowel, just as intussusception might result from the presence of an intestinal tumor at a higher point. In cases of complete prolapsus, a deep sulcus could be felt all around the protruding portion of gut, deeper in the adult than in the child, on account of the greater thickness of the sphincters, and the anatomy of the affection was the same as that of invagination, though the latter name was ordinarily reserved for those cases which occurred at a higher point, such as the neighborhood of the ileo-cæcal valve.

Dr. Allen inferred, inasmuch as the specimen included a portion of the bowel with a complete investment of peritoneum, that the growth had arisen from the upper third of the rectum. This portion closely resembles the colon in its structure as well as in its morbid conditions. Intussusception would be likely to occur here, and not prolapsus. Prolapsus, indeed, would appear to be confined to the middle and lower thirds of the rectum. The initiation of prolapsus is the descent of the mucous membrane and the circular muscular layer away from both the longitudinal muscular layer and the fibrous layer which remains intact. This is the essential feature in the mechanism of prolapsus, no matter how the process may be complicated.

Dr. Ashhurst said that systematic writers distinguished between *partial* and *complete* prolapsus of the rectum, and that while Dr. Allen's description was correct as applied to the former variety, in which the protrusion involved only the mucous and submucous tissues, yet in the *complete* form of the affection, in both adult and child, all the coats of the bowel were implicated, and there was actually an invagination of the rectum through the anus.

Dr. Mears asked if there was present in this case any displacement of the uterus. He thought that the condition found to exist in the rectum might have taken its origin in the constant pressure exerted by a posterior displacement of the uterus, a prolapse of the anterior wall of the bowel thus occurring. This condition of prolapse would be increased by the efforts made to evacuate the contents of the rectum, and eventually form such a tumor as is described to have existed in this case.

He believed that a like condition of prolapse might be occasioned as a result of the use of a large pessary, such as it is stated the patient had worn for a period of twenty years. He agreed with Dr. Ashhurst with regard to the proper designation of the condition, it being one of prolapse and not of intussusception. *December* 13*th*, 1877.

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8. *Perforated appendix vermiformis cæci caused by fecal concretion.*

Presented by Dr. R. G. Curtin.

Through the kindness of Dr. William C. Crooks, I am able to present this specimen. Mr. M., æt. 36, of sober, steady habits, sedentary life, and a wood-carver by occupation. About eighteen months previous to his last illness, he suffered from what the attending physician pronounced to be *dysentery*, bloody and mucous stools being frequent. Several weeks elapsed before he regained sufficient strength to resume work. After recovering from this attack he made no complaint, and appeared to enjoy good health up to ten months ago, when he experienced a severe cramp-like pain in the lower bowel, which was almost instantly relieved by a dose of castor oil and laudanum, although a tenderness remained for a time over the right iliac region, which, however, attracted no special notice.

On the 24th of last month (December), while at light work, he was similarly affected, and in the evening took a dose of castor oil and laudanum. This not relieving him, he called at my office the next morning. Anticipating an attack of pelvic peritonitis, I advised him to go to bed, and directed warm applications to the abdomen, also administered an anodyne, to quiet and relieve pain. Next day when I visited him I found him suffering with a marked pelvic peritonitis. At the end of the fourth day his condition began to improve. A gentle purgative being administered, in connection with an enema of warm water, a discharge of small hardened fæces, followed by a copious discharge of muco-purulent matter,

was effected. Apparent convalescence continued until the evening of the 3d instant, when the pain over the seat of the old soreness, in the right iliac region, accompanied by a small, quick pulse, tympanitic abdomen, cold extremities, and vomiting, became intense. Morphia, chloral, and bromide of potassium in large doses failed to give any relief, and death took place between 3 and 4 A.M.

Autopsy, six hours after death.—On opening the abdominal cavity the omentum and intestines were found matted together with a recently formed lymph (except as noted below). A creamy pus exuded from the interstices of the mass, amounting to perhaps four ounces. The rectum was found to be surrounded by old inflammatory induration. All around the right iliac region the small intestine, and the caput coli, as well as the rectum, showed evidence of old inflammatory trouble, being covered with, and glued together by, old lymph. Over the position of the appendix of the caput coli I separated the small intestine, which was adherent, and as the incarcerated appendix came into view, a small feculent nodule escaped from an ulcerated opening through the wall of the cæcal appendix, about an inch from the distal end. This nodule was one third of an inch in length, and about one eighth of an inch in diameter. It was quite firm at first, and seemed to have a minute white nucleus, but the whole of the pellet softened down when macerated in water. The ulcer freely communicated with the bowel above. Below the ulcer, the veriform appendix was increased in size and very firm: the cavity of this portion was found to be obliterated. The organ was about two and three-fourths inches, which is rather longer than is usual.

Dr. Longstreth said that disease around the appendix vermiformis was not so uncommon as the books would lead us to think, and that from considerable experience on the subject, some involvement of this region in an inflammatory lesion was not very rare, and that, too, without bad results in very many cases. Inflammation can occur at this part, entirely locally limited, and end rapidly by plastic adhesion, as we find, *post-mortem*, is so frequently the case with the pleura; yet in the peritoneum, as in the pleura, there may be no life-history of the occurrence, with the symptoms which are usually considered to indicate an inflammation of a serous membrane. In the one case the attack may be looked on as a colic, in the other it may be thought of as a muscular rheumatic pain. In fact, from the anatomical relation of the two parts to their respective surroundings, it is conceivable that many times local inflammation might occur, and end by plastic adhesion, without involving the peritoneum generally; in the pleura, with its constant rubbing, it is much more difficult of con-

ception how any irritant affecting one part does not rapidly involve the whole in inflammatory changes.

In regard to the question of the occurrence of inflammation remaining undiscovered, nothing is more likely, judged of by *post-mortem* experience; no disease, such was his observation, came more frequently to *autopsy* undiagnosed than peritonitis, and that, too, even when the inflammation is general, of some duration, and of a severe character. The class of cases in which he had met peritonitis, not positively known or even suspected during life, include perihepatitis with cirrhosis, tuberculosis, typhoid fever with or without perforation, and injuries or operations in the abdomen. The reverse of this statement is not an infrequent occurrence, that the diagnosis of peritonitis is not confirmed by the *autopsy;* and this statement does but confirm the general growing opinion that certainty of diagnosis in diseases, especially those leading to inflammation of the peritoneal membrane, is one of the most uncertain problems of the art.

In regard to the question of a necessary connection between the presence of hardened fecal matter in the appendix cæci and inflammation, it was the result of his observation that fecal matter is pretty constantly present within the tube, but that its consistence and amount vary very greatly. In many cases, examined with care, he had found single small hardened masses; in a few instances there was more than one such mass; in one case, which he particularly calls to mind, the appendix was occupied in its entire length by masses having the date-seed form. In none of these cases was there peritonitis, although in some the mucous membrane was thickened.

Dr. Shakespeare said, in respect to the presence of foreign bodies in the appendix vermiformis, that he had himself seen two or three examples, without any inflammatory action occurring, and, so far as he could remember, it appeared to him not rare to find foreign substances in the appendix, when the *post-mortem* had been thorough. Dr. S. also thought it possible for foreign bodies to pass into the appendix and again out into the intestines. He believed that it was even exceptional for small smooth bodies to give rise to serious trouble originating in the appendix.

January 10*th*, 1878.

9. *Abscess of liver, and empyema.*

Presented by Dr. E. T. Bruen.

Joseph Donnelly, æt. 22, single, was admitted to the Philadelphia Hospital November 2d, 1877. He complained, on admission, only of indigestion and malaise, which he had noticed for two weeks. He had always been a healthy man; he had never suffered from syphilis; his occupation had been that of a laborer. His family history was imperfect, as he had not seen his parents for some years.

On examination of his chest, which I made as a routine practice, my attention was arrested by the marked accentuation of the second sound of the heart over the aortic valves. This led me to make a careful examination, and I found there was dulness upon percussion on the right side of the chest in the axillary region, from the sixth rib to the base of the chest, extending anteriorly to within two inches of the nipple-line; beyond this point the dulness usually occasioned by the presence of the liver was noted, which dulness extended, however, nearly an inch and a half below the margin of the ribs; there was no movement of the liver upon forced inspiration. Over the remainder of the anterior portion of the chest good resonance could be obtained. Posteriorly the dulness in the axilla extended to the angle of the scapula; elsewhere there was resonance. The respiratory murmur over the resonant region was feeble; over the dull portions of chest, very faint and distant.

Upon the left side nothing unusual was noted. On the 8th of November, a swelling was observed on the right side, including a space over the seventh, eighth, and ninth ribs, which was painful on pressure. He suffered especially if the ninth or tenth rib was pressed upon from behind, the pain being anteriorly near the position of the swelling. I inferred that the ribs last mentioned were diseased. At this time he commenced to expectorate purulent matter with cough, and his temperature, which had been normal, with the exception of the day of his admission, when it was 102° F., rose to 100° F. The expectoration came up in large amount, several mouthfuls at a time, mostly when he was in the recumbent position. Sometimes he would pass the entire day without expectoration, again would be choked by it.

From the 9th to the 16th, he suffered from hectic and sweating, temperature ranging from 99° F. to 104° F. On the 16th, believing there was a purulent collection in the pleural cavity, or in the liver, an exploring needle of an aspirator was introduced into the ninth interspace at seat of

pain and swelling, selecting this position on account of the doubt in the diagnosis. Simply a bloody fluid was obtained. On the 21st, the needle was again introduced through the eighth interspace, and eight ounces of pus removed, and the cavity injected with a dilute solution of Lugol's iodine, gtt. x to f℥i of water. The external swelling, however, still increased, and the hectic was unchanged.

About the 29th, there appeared to be a circumscribed pneumothorax at the position mentioned above, as amphoric breathing and tympanitic resonance replaced the dulness. On the 29th, therefore, he was etherized, and my colleague, Dr. J. Wm. White, cut down upon the swelling and introduced a rubber drainage-tube in the eighth interspace, at the anterior border of the axillary region, and brought out the tube in the same interspace, about four inches posteriorly. Very little pus was evacuated at the time, for most of it had been previously expectorated through the bronchial tube. The operation was more difficult than is usual, on account of the infiltration of the thoracic wall.

For the following three weeks the hectic was checked, and the patient gained in flesh and strength, becoming able to walk about the ward; the temperature being always below 100° F.; no sweating. Drainage at first took place freely, and for the last two weeks in December in diminished amount. The cavity was injected daily with dilute solution of iodine (Lugol's solution) or carbolized water, coughing never being occasioned unless the solutions were made strong of iodine. He always tasted the substance with which he was injected. The incision into the chest-wall, made to introduce the tube, healed completely, the infiltration disappeared, and he seemed to be doing very well. His appetite and digestion also improved decidedly, but occasionally he would suffer from attacks of hiccough. After the 17th of December, however, his temperature rose every night to 101° F., and he ceased to improve.

January 1st, he passed out of my hands, as my term of office expired, and he died of acute pleurisy on January 6th, 1878.

Post-mortem examination.—Upon opening the chest, the right pleural cavity was found to be completely filled with a recent pleuritic effusion, sero-fibrinous in character, the lung occupying the lower portion of the pleural cavity, near the diaphragm and vertebral column, and flattened to about the size of a man's hand. It was bound down in this position by adhesions. The fifth and sixth, eighth, ninth, and tenth ribs were found to be diseased; the chest-wall over the lower ribs was very much thickened, and contained a small amount of pus, which was confined by adhesions between the liver and the diaphragm. An opening through the diaphragm

allowed this pus to communicate with the lung or the pleural cavity. The drainage-tube was found at this place surrounded by the lung, which had been compressed upon it by the recent pleurisy, the compression being aided by adhesions. In the upper part of the right lobe of the liver there was an abscess about the size of an orange, the cavity of the abscess being distended with contained pus, and which communicated with the abscess in the thoracic wall by an opening which was so small and tortuous that the abscess did not empty itself. The remainder of the liver-tissue was soft and fatty. The intestinal canal was examined throughout, but found healthy. The kidneys were nearly normal, though slightly fatty.

Remarks.—The improvement of the patient's condition after the drainage-tube had been introduced is worthy of attention. The hectic for several weeks was totally checked. The cavity appeared to grow smaller during the treatment by injections, for at the time of the operation a probe could be moved about readily in different directions. As previously stated, an hepatic abscess was suspected at first. The even range of the temperature for so long a time was favorable to this hypothesis. Dr. Murchison states that cases of hepatic abscess are often mistaken for remittent fever on this account.

The liver itself, though extending an inch and a half below the ribs, was stationary when a full inspiration was taken, which Frerichs considers an important evidence of empyema, as in abscess of the liver the descent of the diaphragm on inspiration will depress the liver. The physical signs and expectoration appeared to indicate an empyema with perforation into a bronchial tube.

The disease of the ribs I always suspected was the cause of the empyema, but from the absence of any history of local injury, or of syphilis, I could not feel sure the disease was not secondary. In the light of the *post-mortem*, it appears to me the sequence of these lesions was first the abscess in the thoracic wall, which caused, by extension of inflammation, an abscess in the liver, which abscess subsequently perforated the diaphragm and pulmonary tissue and was expectorated through the bronchial tube, the empyema being a separate lesion, or else the empyema resulted from the perforation of the diaphragm, really being a part of the first abscess.

I have read recently some interesting cases in the *London Lancet* for November 17th, 1877, reported by Dr. Fenwick, of abscess of the liver perforating the diaphragm, and being expectorated, with symptoms very similar to this case.

There is one other consideration in reference to the abscess which it is only right should be stated, viz., the possibility that at the time of the first

puncture, when I obtained only blood, the liver was penetrated. But I claim that the operation, even with this risk, was justifiable, because there was distinct fluctuation where the needle was introduced. If the abscess in the liver had been reached by the aspirator, a cure might have resulted.

Dr. Guitéras said that this case was admitted to the Philadelphia Hospital soon after the death of the case of abscess of the liver, presented by him to the Society some weeks ago. For a time the temperature of Dr. Bruen's patient ran a remarkably regular course, like to a malarial fever. This, you may remember, was a marked feature of his own case. In the present instance he never gave up the diagnosis of abscess of the liver, though the symptoms became subsequently masked by the disease of the ribs and of the pleura. The very low position of the pain and tenderness also pointed to abscess of the liver. It would have been interesting to study this case in the light of the observations made by Fenwick (*London Lancet*, November 17th, 1877). He reports several cases where liver-substance was found in the pus discharged from abscesses of that organ. Liquefying the pus by adding a little ammonia, he found in the sediment the liver-cells more or less disintegrated.

Some time ago Dr. Guitéras reported to the Society that since July of 1873 there had been in the men's medical wards of the Philadelphia Hospital eighteen deaths from empyema; that in every one of these cases the operation of paracentesis had been performed more than once; that in some of them different operative procedures had been tried; that in the great majority of them the effusions, primarily serous, had been converted into purulent by the operation; that, excluding the operations for passive effusions, these eighteen cases included the great majority of those operated upon within this period, since the most careful search failed to find more than three successful results; one was of traumatic origin, the other was a comparatively recent effusion, and the third he knew nothing about. In these three cases the effusion never became purulent.

Within the last eight months the operation has been performed in two cases; this is the second one; the first also terminated fatally; a third one is now at the point of death in his wards of the hospital (and has since died).

He did not think the atmosphere of the Philadelphia Hospital was a bad one. With a population of nearly 4000 in the whole institution, it may be said that septic diseases do not prevail there; diphtheria never; typhoid fever very seldom originates in the house; and when introduced, they do not tend to spread. He thought pyæmic infection was not frequent

in the surgical wards. An obstetrical department is out of place in an institution of this kind, and it is no wonder that septicæmia prevails there. Our patients do not get the best of diet, and they are frequently men broken down by excesses; yet the influences of these degradations of the constitution are not shown to such a fearful extent in other diseases.

This operation is a rational one. He knew it had been very successful in the hands of some, specially Dieulafoy, who has given particular attention to all the details; but, as usually performed, it needs perfecting, and we will never perfect it if we deceive ourselves as to its dangers.

Dr. Starr said that, in regard to the results of the operation of paracentesis thoracis, the experience of Dr. Guitéras in the wards of the Philadelphia Hospital did not correspond with his own at the Episcopal Hospital, since, so far as he remembered, nearly all of quite a number of operations performed during his connection with the latter institution had been successful; in some instances a permanent cure resulting, while in other complicated cases, where no termination but death could have been expected, great relief was afforded, and life unquestionably much prolonged. Not to multiply examples, during his last term of service several patients were subjected to the operation. From one case of acute pleurisy, with effusion in the left pleural cavity, a considerable quantity of serous fluid was withdrawn; the man's condition improved rapidly, and he left his bed convalescent in ten days. When discharged, a month afterwards, there were no evidences of fluid in the pleural sac, nor were there any six weeks later, when he visited Dr. Starr at his office, although he had undergone much exposure in the interval.

The second patient had been tapped upon several occasions for empyema of the left side, and had worn a drainage-tube. When admitted to the hospital he was very weak and emaciated, and suffered from hectic fever, profuse night-sweats, and cough; there was a slight purulent discharge from an opening in the seventh intercostal space, through which the drainage-tube had been passed, and upon introducing a small flexible catheter about six ounces of pus, having a faint disagreeable odor, flowed away. For some time the pleural cavity was washed out through this opening, without much improvement in either the local or constitutional condition. Subsequently a free opening was made into the pleural cavity in the eighth interspace, a drainage-tube introduced, and the cavity washed out daily with a weak solution of carbolic acid. After this the improvement was rapid, and at the present time there is little or no formation of pus, no hectic fever, night-sweats, or cough, and the patient has gained more than fifteen pounds in weight, and feels well and strong enough to

work. Here, of course, the ultimate result is still doubtful, but the prospect is bright.

In a third case, one of pneumo-pyothorax occurring in the course of phthisis, the left chest was punctured twice, and a large quantity of pus evacuated: first operation, November 18th, thirty-six ounces; second, December 18th, sixty ounces. In this case the operations were undertaken to relieve distressing symptoms, and were more than satisfactory in their results. He thought, therefore, that a sweeping condemnation of the operation was unjustifiable.

Dr. Longstreth thought that diagnosis of abscess of the liver, by examination of the pus during life, was very uncertain. There was a case some time since in the Pennsylvania Hospital which presented the symptoms of an abscess of the liver. The pus was drawn and examined, but from the character of the cells no conclusions could be determined upon. Dr. L. did not think the method of Fenwick could be borne out, since in an abscess of the thigh we do not find muscular fibre. With bone it is different, as we find calcareous particles; but we cannot say they are bone-cells.

Dr. Guitéras said he did not think any doubt could be thrown upon the value of these observations. Fenwick goes even so far as to attach prognostic value to them. When the liver-cells are found in abundance, the breaking down of liver-substance is progressing, and the prognosis is unfavorable; when they are absent or very much disintegrated, he has found the result more favorable. In a case of suppuration of a cyst, he only found the calcified remnants of hydatids.

Those cases in which a spontaneous opening occurred externally, he believed, do better than those operated upon. Whilst this fearful mortality has been going on in the wards of the Philadelphia Hospital, he has had under observation, in the same wards, for four years, a case of empyema with an external fistula which died a few weeks ago of pericarditis, with pneumonia of the healthy side.

The operation is more favorable in children, probably on account of the elasticity of the chest-walls. All the cases he had reported were adults, and their histories were followed out to the end. Unfortunately, this is not always done, and many incomplete histories are published as cures. He believed also that in this, more than in any other serious operation, the unfavorable results did not reach the light.

February 14*th*, 1878.

10. *Hepatic abscess discharging through the right lung.*

Presented by Dr. Louis Starr.

I am indebted to Dr. Forbes for the privilege of presenting these specimens, the patient from whom they were taken having been under his care in the Episcopal Hospital for a short time prior to death. They were obtained from the same case from which Dr. Packard removed the carcinomatous tumor which was exhibited to the Society on November 22d, 1877,

The patient was a man 58 years of age, who was admitted to the surgical ward of the Episcopal Hospital on October 26th, 1877. The tumor referred to was removed from the parotid region on October 29th. On November 4th he was attacked with pneumonia of the lower lobe of the right lung. The acute symptoms began to subside in about two weeks, but he continued to have considerable cough, attended with the expectoration of somewhat tenacious, blood-stained mucus. Three weeks before death, which occurred on January 21st, 1878, the expectoration changed in character, becoming more abundant, purulent, and slightly offensive in odor.

The *autopsy* was made twenty-four hours after death. On opening the thoracic cavity, the surface of the lower third of the right lung was found to be bound to the chest-wall by moderately-firm pleuritic adhesions, while the rest of the right pleural cavity was filled with a reddish sero-purulent fluid. In the upper postero-lateral portion of the fissure, between the superior and inferior lobes, there was an abscess an inch and a half in diameter, partially filled with thick purulent matter, and lined with a firm membrane. This abscess did not involve the lung-tissue, but communicated with the pleural cavity through an opening one-fourth of an inch in diameter, having thin, well-defined edges. In the lower part of the middle lobe there was a large cavity, with very irregular walls, having a free communication with one of the larger divisions of the right bronchial tube, and also, through a perforation in the diaphragm, with an extensive superficial abscess, situated upon the posterior portion of the convex surface of the right lobe of the liver. There was some congestion of the middle and lower lobes.

The left lung contained several isolated collections of caseous material, but in other respects was normal.

The abscess of the liver, though not extending deeply into the substance of the organ, measured nearly four inches in transverse diameter. The remainder of the hepatic structure was apparently healthy.

The cardiac walls were flabby, the kidneys were large and fatty, and the spleen was nearly twice its natural size. *February 28th*, 1878.

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11. *Case of diabetes with fatty diarrhœa.*

Presented by Dr. M. Longstreth, for Dr. H. E. Goodman.

S. M. Z., M.D., æt. 55 years, entered the army in 1861 in excellent health, weight about 190 pounds. In the summer of 1862 had an attack of jaundice, diarrhœa, cough, with fever, known as typho-malarial fever; recovered; had a return of diarrhœa, fall of 1863, at Lookout Mountain, for which obtained leave of absence; again had another attack after Sherman's march, for which granted leave of absence from Savannah, Ga., January, 1865. While at Gettysburg, July, 1863, met with an accident by his horse falling on him; supposed injury of spine, causing severe pain in back and limbs, for which he was sent to the general hospital at Georgetown, D. C. After the fall he was subject to pain in the back and numbness in limbs. Wife alleges he had a very severe attack of jaundice and diarrhœa after the war, during which he was not expected to live.

I knew him from 1861 until his death in 1876, and saw him frequently in the field and after the war. From about 1867 or 1868 observed his gradual failing health. October 16th, 1873, was first called to see him professionally. Found him emaciated; weight 125 to 130 pounds; diarrhœa so incessant as to confine him to bed; passage involuntary, of the consistence of sweet oil, leaving greasy spots on his bed-clothing. He alleged he passed two gallons of water daily. Had no fever nor pain, only prostration.

October 16th, obtained specimen of fæces and urine, which Dr. R. M. Bertolet kindly reported, on October 18th, 1873, as follows:

"Dear Doctor,—The urine has a sp. gr. of 1044, an acid reaction, and is devoid of sediment. The normal contents, urohæmatics, urates, uric acid, diminished; chlorides normal in amount; triple phosphates, earthy phosphates, sulphates, apparently diminished quantity.

"*Abnormal contents*, glucous very abundant; no traces of albumen; sulphate of copper, etc., very readily oxidized. The fæces contain mucus, epithelial cells in various stages of disintegration, and foreign matter; but the great bulk composed of fat, in the form of spherical masses, composed

of dense aggregations of minute crystals, such as are presented by margarine. It is readily soluble upon the addition of chloroform under the glass cover.

"Very truly yours,

"R. M. Bertolet.

"113 S. Broad Street."

As the case was one of unusual interest, I desired Dr. Da Costa in consultation. No tumor was felt in the abdomen. He was placed on a diet in which starch and fatty matters were excluded; at first skimmed milk, diabetic-bread, pancreatin, and pepsin, with quinine and opium in small quantities. Under this treatment he gained flesh, the diarrhœa ceased, no fat was observable in the fæces, and the sugar diminished in the urine. Whenever he would transgress in his diet, the diarrhœa and fatty discharges would return, and the urine increase in quantity. I looked after him for about a year, during which he attended to his business. He afterwards fell into the hands of his old classmate, Dr. McClintock, who continued to treat him until he died, in June, 1876. I saw him while visiting his son for a deformity of the ankle, about a month or two before he died, and found him dropsical and very feeble.

He desired his wife to have me make a *post-mortem* examination, which Dr. J. Morris Longstreth kindly performed for me twenty-four hours after death.

Autopsy, June, 1876.—Z., æt. 55. Thirty-nine hours after death. Body in ice. Emaciated.

Cavities.—*Abdomen* contained a small amount of serum, but showed no trace of recent inflammatory adhesions.

Right pleura.—The sac was obliterated by firm adhesion of the upper lobe, in which were found numerous ecchymotic patches; the lower part contained a collection of serum and a mass of recent flocculent lymph.

Left pleura.—There were a few old adhesions of the *left lung* to the chest-wall, a small collection of serum, but no recent lymph. The *pericardium* contained f℥ij of serum, with a little flocculent lymph.

Heart was covered with a small amount of fat. Left ventricle hypertrophied. Aortic semilunar valves showed no marked change. The orifice was rigid from atheromatous changes (one place was calcareous) just above the valves. The hydrostatic test showed slight incompetence at this orifice. Mitral valve was atheromatous in places.

Lungs.—The *left* was crepitant throughout, much congested posteriorly; its upper lobe normal; lower lobe on section exudes abundance of bloody,

slightly frothy serum; its tissue unaltered in consistence. The *right* upper lobe showed a number of cheesy masses, and one cavity the size of a pullet's egg, containing thick, yellowish-white matter. Its lower lobe was to a great extent carnified (compressed by inflammatory exudation), showing no cheesy masses, and no evidences of pneumonic consolidation.

Spleen was enveloped in a complete fibrous (inflammatory) capsule, was large (six inches in length), pretty firm, regular in outline; on section, its tissue was found normal in appearance.

Kidneys.—The left was much congested, the surface uneven and mottled, the capsule decidedly adherent. On section, the vessels were very conspicuous; the relation of cortex to medulla was normal. The whole organ was very considerably enlarged. The *right* presented about the same appearance. It was less congested, and its capsule was less adherent.

Suprarenal capsules were normal in size and consistence. The medullary portion was perhaps rather increased in amount.

On the left side of the vertebral column, behind and below the stomach, was found a rounded, firm mass, about the size of a large orange. It was tightly adherent posteriorly, especially to the connective tissue above the left kidney. The mass was carefully dissected out and found to be an enlargement of the tail of the pancreas. It was a cyst, with firm, rigid walls, so rigid that it did not collapse, nor could even be much indented, although its contents had escaped during the removal, at a point where the wall was thin and adherent. Unfortunately, the fluid contents suddenly escaped, and none could be collected for examination. It was of a yellowish-green color, more fluid in consistence than white of egg. The tumor, together with the rest of pancreas, and about six inches of the duodenum were removed together, ligatures being placed on the intestine before its division.

Subsequently, dissection showed that the cavity of the cyst had no communication with the pancreatic duct. The interior was covered by dirty brownish material, thickly gelatinous in consistence, which could be readily rubbed or scraped off, but had no tendency to separate of itself. Parts of this material were deeply colored black. Within the cyst, attached to the right wall, was found a rounded mass the size of a large plum, having a short fibrous pedicle. This mass was freely movable by its pedicle. The *pancreatic duct* was traced from the intestine through the length of the organ as far as the cyst-wall, at which point it ended abruptly. The lumen of the duct was very large throughout the whole length of the body of the pancreas, and suffered but little diminution as it approached the cyst. The portion of the duct passing in or behind the head of the organ

to reach the intestine was narrow, perhaps narrower than normal; the probe found difficulty in passing this portion of the duct as it approached the wall of the intestine. The duct was opened at the point where it passed from the head to the body of the pancreas, and here were seen two small sacs or pouches in which some calculous matter rested. The glandular tissue of the organ appeared to be entirely atrophied; the head of the pancreas was represented by a lump of fibrous tissue, which was continuous with a mass of connective tissue (new growth) between the stomach, pancreas, duodenum, and transverse colon. This connective tissue surrounded the blood-vessels in this region, and received numerous small branches from them, and was evidently constricting and pressing upon the duct.

The *common gall-duct*, which was also involved in the mass, was somewhat dilated; its entrance into the intestine, whilst somewhat narrowed, was less interfered with than the pancreatic duct.

The *splenic artery* was found passing along the border of the atrophied pancreas, and then on the posterior wall of the cyst, to which it was adherent. At the point where this vessel reached the cyst-wall it gave off a moderately large branch, passing apparently to the cyst, close to the attachment of the pedicellated mass within the cavity. No connection could be traced between them.

The vena portarum and the termination of its principal branches were lying in looser parts of the connective tissue, and seemed not to be interfered with by it.

The *liver* was large; its outline pretty regular; its capsule marked by occasional white patches or spots. On the surface of right lobe was a small spot of atrophy (racemose). On section, its tissue appeared normal.

The *gall-bladder* contained golden-colored bile. In it were found two small biliary concretions. Its mucous membrane appeared normal. Its duct, as well as the hepatic duct, appeared wide. They showed no point of constriction, and no evidence of inflammatory change. The connective tissue, from the transverse fissure of the liver downwards, enclosing the bile-ducts and vessels, was greatly increased in amount.

The *stomach* was contracted. Its mucous membrane showed very considerable *post-mortem* change, but nothing abnormal.

The microscopic examination of the contents of the intestinal tube showed nothing especial. There were present starch granules, partly disintegrated muscular fibres, very numerous oil globules, and amorphous granular matter.

The cortex of the kidney shows, microscopically, fatty changes in the

epithelium of the tubules. At the periphery of the organ there is an increase of connective tissue, especially seen directly beneath the capsule, but also in the contiguous deeper parts. *February 28th*, 1878.

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12. *Scirrhus cancer of the pylorus.*

Presented by Dr. William H. Warder.

Miss Amelia P., aged 35 years, was always remarkably healthy until March, 1877, when she began to suffer from attacks of indigestion. These attacks, which were supposed to be the result of some error in diet, grew worse and more frequent, until there was a constant uncomfortable feeling in the stomach. Domestic, homœopathic, and patent medicines were tried without benefit until July, when she consulted Dr. Conner, who has kindly given me her symptoms at that time and during his attendance. There was languor and debility, sleepiness, uneasiness in the epigastric region, especially after eating; vomiting also occurred occasionally a few hours after meals, of undigested or partly digested food and mucus. The patient was somewhat anæmic. Bowels constipated; tongue moist, but slightly coated in the centre. From this time until December there was no marked change. She was alternately better and worse. Now she began to complain of sour stomach, acid and watery eructations, and heartburn. Anæmia and debility becoming more decidedly marked; countenance cachectic; bowels constipated. The distress in the stomach was of a gnawing character, and described as a "misery." She never admitted having pain of a sharp lancinating character. At this time there was some tenderness in the lower part of the right epigastrium, which was entirely relieved by a blister. Strict attention had been paid to the diet. Oleaginous substances, pure cream, cod-liver oil, essence of beef, were acceptable to the stomach, and produced only a minimum of gastric uneasiness. Saccharine or starchy food always produced a great deal of distress. Cod-liver oil taken in September improved her condition, and she gained about three pounds in flesh. December 13th, passed a restless night, and early on the morning of the 14th vomited about one quart of a dark fluid having a sooty or coffee-grounds appearance; also a small quantity in the afternoon. This was the only time this kind of vomiting occurred during her illness. The stools the day following were much darker than usual, and the breath for eight or ten days subsequently was fetid.

Immediately preceding this black vomiting the patient complained of

intense burning in the stomach, remarking that it felt like a coal of fire. This pain disappeared after the vomiting, and did not return. Dr. Conner pronounced the case cancer of the pylorus.

I saw the patient on February 13th. She was very much emaciated and suffering from great debility; countenance cachectic. Did not complain of any pain in the stomach. No nausea or vomiting. No pain upon pressure over any part of the abdominal walls. She was able to take one and a half to two pints of milk per day, and the essence of a pound of beef, without inconvenience. Did not complain of any pain after eating. She complained only of weakness.

There was no tumor of any kind in the epigastric region. Upon a very careful examination, I found a very small, hard nodule, about the size of a walnut, on a line with the umbilicus, and about one inch and a half from it. This could be handled without giving the patient pain, but did not seem to be very movable. It appeared to be in the region of the head of the pancreas. The bowels were loose and inclined to diarrhœa. The urine was normal in quantity and quality. The temperature 99° F. to 99½° F. in the axilla.

Dr. William Pepper saw the case with me on the 7th of March, and after a careful examination was disposed to think the disease cancer of the head of the pancreas. The character of the symptoms negatived the idea of any considerable degree of pyloric obstruction, though from the occasional attacks of marked gastric disturbance which had occurred at an earlier period of the case, it seemed not improbable that the diseased mass was attached to or partly involved the walls of the stomach. There were no especial symptoms of any kind after this. She simply complained of weakness, and died on the 16th of March.

Post-mortem.—Stomach very much distended, with about one quart of dark, sooty fluid in it. The pyloric portion was on a line with the umbilicus, and about one and a half inches to its right. The fundus or larger tuberosity was resting on the upper portion of the left iliac region. It was healthy, except at the pyloric orifice, where there was a hard nodular mass. Upon opening the stomach there was found an ulcerating surface corresponding to the hard indurated mass. No glandular involvement; liver normal; also pancreas and spleen.

Dr. Pepper said he had seen the case once in consultation with Dr. Warder, and it seemed to him to present much interest, both clinical and pathological. It was a good illustration of the difficulty that often attends the diagnosis of diseases of those organs whose functions are not well known or are with difficulty investigated during life. In the present case

the progress of the symptoms made it clear that malignant disease existed, and careful examination revealed the presence of a small ovoid tumor in the position described by Dr. Warder, a little to the right of the median line and about one inch above the line of the umbilicus. The diagnosis chiefly rested between pyloric or pancreatic scirrhus. It must be remembered that at no time had there been symptoms of pyloric obstruction. Vomiting had occurred several times in the course of a few months, and some kinds of food caused distress; but as a rule the patient was able to take and digest a fair amount of nutritious food. In some cases of pyloric cancer symptoms of obstruction, distention of stomach, vomiting, constipation, disappear late in the course of the disease, owing to the occurrence of ulceration; but here no such symptoms occurred at any time. It is true that at the *post-mortem* the stomach was found markedly enlarged; but this could not be detected when I saw the patient. The belly was retracted, and there was dulness on percussion everywhere, save just below the ribs. This was due to the fact that the intestines contained little gas, while the stomach contained scarcely any, the only contents being a considerable quantity of grumous fluid which gravitated to the greater curvature. The tumor was unusually low down for pyloric scirrhus; and it also seemed less movable and more closely attached to the vertebral column than is usual with such growths. The above points seemed certainly in favor of the seat of disease being the head of the pancreas. Considering the quantity of food taken and retained, and the absence of fever or severe pain or any wasting discharge, the emaciation was rather remarkable. It could not be said that there was any unusual proportion of fat in the stools; and the patient was too ill to justify the administration of oil with the view of ascertaining the power of digesting fat. On the other hand, various symptoms met with in a large proportion of cases of pancreatic cancer were absent. Among these may be mentioned pain in the back, and jaundice. Upon the whole, however, it seemed most probable that the scirrhus nodule was seated in the head of the pancreas, and that the pyloric end of the stomach was closely adherent or even involved. It has been seen, however, that this view was not confirmed by the *post-mortem* examination; although even now it seems difficult to see how a different opinion could have been formed. It is finally interesting to note that, although symptoms of pyloric obstruction were absent, there is a certain degree of contraction of that orifice. Thus, the little finger can be passed through it only a little beyond the first joint. *March 28th*, 1878.

13. *Cancer of the ascending colon.*

Presented by R. G. Curtin.

John McL., æt. 64 years, married, native of Scotland.

The patient, who was by occupation a stone-cutter, came under observation on the 15th of January, 1877.

When he was 21 years old, he had an attack similar to the one for which he came under treatment last January, except that he did not then notice any tumor at the seat of pain. This seizure passed by, but kept him so reduced that he was for a time unable to work.

In the early part of 1876 he had some rheumatic symptoms in the legs, and suffered from weakness and vertigo.

With these exceptions the patient stated that he had been perfectly well up to within eight months of the time he applied for treatment.

In the month of June, 1876, while lifting a heavy stone, he suddenly felt a stinging or *jagging* sensation, of a warm or burning character, in the right iliac fossa, which continued for two weeks, sufficiently severe to unfit him for work.

About two months after this lift, his attention was directed for the first time to a tumefaction in the right iliac fossa, at the point in which he had felt the sensation of pain. This tumor was somewhat tender on pressure, becoming more so as it increased in size. The patient compared the pain to that of an abscess, as if matter were present.

In August he had a great tendency to nausea, and frequently vomited. For a month or two only he belched up "wind and slime." When swallowing food he noticed an inclination to eructation. He would feel the gas working up and down, and obtained temporary relief after belching.

Never passed blood with his stools. Bowels were regularly opened until about October, 1876, when first symptoms of constipation appeared.

He would go to stool with flatulent colicky pains, and fail to be relieved. Suffered much from *rumbling* in the bowels, accompanied with pain.

When first seen, in January, 1877, he had coughed for two months, expectorating about a tablespoonful in twenty-four hours.

Auscultation revealed, on the left side, noisy and harsh inspiration, with audible and prolonged expiration, about the mammary region; on the right side in the corresponding region harsh and noisy inspiration, with expiration same as on the left side.

There was progressive loss of flesh and strength, and the patient died,

on the 18th of May (last), of exhaustion and starvation. There was extreme emaciation.

Post-mortem.—On opening the abdomen, a tumor, the size of the fist, was found about one-third of the way up the ascending colon. The only evidence existing of peritonitis was a small patch outside the tumor, where a loop of the upper part of the ascending colon was bent down, and attached to the tumor. The tumor was hard, and on section the tissue grated under the knife like cartilage. The inside of the tumor was in an ulcerated state, with patches of red, pulpy mucous membrane scattered here and there. The alimentary canal was patulous through the tumor, but very much constricted.

The difficulty in making a diagnosis in this case from chronic perityphlitis is apparent from several considerations. First, he had stated that he had had a soreness, with evidence of inflammation, in the same place when a boy. Second, the soreness came on suddenly after a heavy lift. The growth was very slow, and ceased a year before death.

A daughter of this man had an ulcerated condition of the small intestine, seated near the same place, two years ago. The specimen was presented to this Society.

Dr. Seiler made a microscopic examination of the tumor, and found the muscular tissue of the intestine, at the place of the new formation, to have disappeared; in its place a connective tissue forming alveoli, in which were epithelial cells, was found.

Dr. Tyson said in reference to one of the symptoms mentioned by Dr. Curtin, the sensation "as if something had suddenly given way" within the abdominal cavity, that he thought this symptom was due here to the rupture of an adhesion, which had formed at a previous period, when inflammation first set in, due to the irritation of the growing mass. Some years ago he had met the same symptom in the case of a young man, who, while gunning, felt something give way in the abdomen as he made a long leap; immediately peritonitis was developed, and he died promptly.

June 27th, 1878.

III.—THE VASCULAR SYSTEM.

1. *Fatty heart, with polypus of right auricle.*

By Dr. Frederick P. Henry.

Rachel N., æt. about 50, was admitted to the Episcopal Hospital on October —. Her clinical history previous to admission was very meagre in details, and is essentially comprised in the fact that her illness began in December last with dyspnœa and precordial distress, and gradually increased in severity. The date of the beginning of the dropsy—the most prominent symptom—was not obtained.

On admission, she was extremely feeble and very anæmic-looking, but did not present an appearance of cyanosis. Her feet and legs were quite œdematous, and the abdomen greatly distended with fluid: at the widest portion it measured forty-eight inches.

I examined the urine carefully, but found neither albumen nor casts, The dyspnœa was not great when in the sitting posture, due doubtless to diminished sensibility of the respiratory centre from carbonic-acid poisoning, for she was very drowsy. A portion of each night had to be passed in a chair.

The symptoms relating to the heart were all objective, the patient making no complaint, and were very interesting. The pulse combined, in their highest degrees, the qualities of frequency, irregularity, and inequality. Several feeble beats would occur in rapid succession, to be followed by two or more pulsations more normal in fulness and rhythm, then perhaps an intermission equal in time to one or more of the most natural pulsations, and again several rapid, fluttering beats. During another fraction of a minute this order might be entirely changed. The action of the heart, it is important to remark, entirely corresponded with the radial pulse. It is well known that we may have a pulse similar to the one I have described with a regularly-acting heart,—the "*pulsus paradoxus*" of Kussmaul,—or, on the other hand, we may have a regular pulse with an irregularly-acting heart, *e.g.*, when the ventricular contractions are not synchronous. The heart-sounds were faint, but uncomplicated with murmur, and the apex-beat was feebly felt in the normal

situation. Owing to the great weakness of the patient, the examination was not protracted, and no attempt was made to determine the area of precordial dulness; as will be seen later, the condition of the adjacent lung would have rendered any such attempt hopeless.

I had previously seen several hospital cases precisely similar in every respect to the one under discussion, in none of which did I obtain an *autopsy*, for the reason that none of them died while under my care; indeed, the tenacity with which they clung to life was always a source of wonder to me. My diagnosis in all these cases was atheroma of the orifices of the coronary arteries, with subsequent fatty degeneration, and, as it is now confirmed by an *autopsy* in one case, I have no doubt whatever that it was correct in every instance. Ordinary fatty degeneration does not produce more than a slight irregularity, and even that exceptionally, whereas the combination with atheroma of the coronary orifices produces both it, and inequality in a high degree. The scanty and irregular supply of blood, it is easy to understand, would produce just such an action, whether we regard the cardiac contraction as dependent upon a direct influence from the blood or an indirect one through the cardiac ganglia.

The patient died suddenly on October —. Tapping was not resorted to, because the effusion diminished very preceptibly under a treatment designed to promote arterial tension and increase the action of the bowels.

At the *autopsy*, the pericardial sac contained about two ounces of a greenish-yellow serum, the sac itself being healthy. The heart was considerably enlarged by dilatation; its walls pale, flabby, and evidently fatty. The auriculo-ventricular valves, on both sides, were thickened, especially at their free borders, and had lost their transparency, being of an opaque yellowish-white. Were it not that they appeared perfectly competent, I should say, from their appearance, that they were the seat of a chronic endocarditis. The question of competency is not always easy to determine, but I think it will be agreed by those who examine these valves that, if the amount of thickening present were due to inflammation, there would also be present an amount of induration and contraction that would produce very manifest stenosis, or insufficiency, or both. If any abnormality were present, it was undoubtedly insufficiency, and was probably relative owing to dilatation of the venous ostia.

If this thickening is not inflammatory, what is it? I believe it to be fat,—a primary deposit of fat,—not atheroma, which is a fatty degeneration of previous inflammatory deposit; and think I have more than once observed fat in this situation that has been mistaken for atheroma. Fat in this situation is, so far as I know, not mentioned as a cause of cardiac

disease, and, unless it be excessive, could hardly be so considered; but the limits to its deposit here, as in other situations, are probably very indefinite.

Can we decide this to be fat without the microscope? I think so, for the reason before given, that were the amount of thickening present due to inflammation, contraction would not be absent. The contraction of inflammatory deposits in this situation is peculiarly great.

In the next place, can the exact anatomical seat of this deposit be determined without the microscope? Let it be observed that the degree of this fatty thickening in the two auriculo-ventricular valves is very different, being much greater in the mitral than in the tricuspid. Now, unless our views as to the deposit of fat and fatty degeneration are entirely wrong, this is just the contrary of what we ought to find. The presence of highly-oxygenated blood is inimical to the deposit of fat, and in the left auricle we have the blood charged with oxygen to its utmost capacity, while in the right auricle the conditions are entirely reversed. Is this therefore a deposit of fat, or a degeneration—a metamorphosis—of elements existing in both valves in an unequal degree? In the mitral valve it is generally conceded that muscular fibres are present; indeed, I do not know that it is called in question by any one who has examined the subject; while in the tricuspid they are comparatively so rare, that certain authorities are quoted in the text-books in support of their existence. They exist, therefore, in a very unequal degree on the two sides, so that a fatty degeneration on both sides of these valvular fibres would produce such dissimilar appearances as are observed in this specimen. Another interesting question upon which greater light might be thrown by further study of fatty changes in these valves is this: in fatty metamorphosis is there increased bulk of the diseased cell? If my views are correct, this must be answered in the affirmative, for the valves are thickened, and it would not be difficult to find numerous facts in support of this conclusion. At present I need only refer to the liver.

The right auricle contains a tumor which, under the name of cardiac polypus, was a source of great interest to the older anatomists. It is a cyst about the size of a filbert, containing a puriform fluid, and is firmly attached to the endocardium. It is unnecessary to describe the mode of formation of these cysts, but I will call attention to the fact that they may be the cause of serious functional disorder, and, apart from the formation of embolism by the discharge of their contents or their detachment, may, by their interference, produce dyspnœa, dropsy, and sudden death. This is admitted by Schroetter (Ziemssen, vol. vi.). In the present case the

polypus is probably secondary to the fatty degeneration, to which was owing the feeble circulation requisite to its formation.

The liver was rather below the normal size, lighter in color and denser than normal. On section, it presented the nutmeg appearance in a marked degree. There was probably a combination of red atrophy with increased formation of interlobular connective tissue, cirrhosis. This cirrhotic complication was a principal factor in the production of the ascites. I make this assertion *because the urine did not contain albumen.* In my experience, an amount of cardiac disease sufficient *in itself* to produce ascites, is invariably accompanied with albuminuria. There may be exceptions to this rule, which is, as I said, based upon my own experience solely; but at least it is not too much to say that in cardiac disease with ascites, but without albuminuria, cirrhosis of the liver is to be suspected, and the cirrhosis, in relation to the heart disease, may be either primary or secondary; in the former case, however, without there being any causative connection.

The kidneys were greatly congested and decidedly granular.

The lungs were healthy, with the exception of the bases on both sides, in which there existed hypostatic pneumonia. The tongue-shaped process of the left lung, and a portion of the anterior border of the superior lobe, were also consolidated, showing that any attempt to map out the area of precordial dulness would have been futile. The larger bronchi were congested; otherwise the lungs were perfectly healthy.

The preceding remarks may be summed up in the four following propositions:

(I.) The auriculo-ventricular valves contain muscular fibres whose physiological action is not thoroughly understood, as is evidenced by the numerous theories concerning the mechanism of their closure.

(II.) These fibres undergo fatty degeneration in common with the myocardium, and perhaps separately, and thus whatever active function they possess may be impaired or lost.

(III.) In addition to such loss of function, fatty valvular degeneration produces a mechanical interference through thickening of the leaflets.

(IV.) Thickening and opacity of valves, without contraction, is to be referred to fatty deposit or metamorphosis.

Dr. Tyson thought it more reasonable to suppose the fatty change in the valves to have been secondary to an inflammation and proliferation, succeeded by a fatty degeneration of the proliferated cells. The contraction of the valves, referred to by Dr. Henry as absent, he thought not a necessary consequence of inflammation. Dr. T. would expect it only in extreme degrees of inflammation. In regard to determining whether the fatty

change was primary or the result of inflammation, Dr. T. thought we might be aided by the presence of unaltered proliferated cells alongside of others, the seat of fatty change, and alongside of these also the products of complete disintegration, including cholesterin plates.

Dr. Nancrede thought it possible to have a fatty change without inflammation, due to imperfect nutrition of the parts.

Dr. Guitéras said that the lesions in this case seemed readily explicable in the usual manner. These valves are not in a passive condition favoring a fatty infiltration. He thought that the case was one of commencing general atheroma, which, together with the disease of the kidneys (cirrhosis), subjected these valves to an unusual strain, giving rise to a slow inflammatory process. This explains why the left heart is affected long before the right, because the former is subject to the obstructions in the general circulation. He had frequently seen just such valves in the early stages of atheroma, and thought that contraction would generally follow in later stages. Dr. G. said that he would remind the Society of the interesting article on "Atheroma" written by Fothergill for the *Philadelphia Medical Times*, where he uses with great advantage the well-known theory of increased vascular tension, to explain the lesions of the heart in general atheroma.

Dr. Henry replied that there are in this case very slight atheromatous changes in the aorta, chiefly about the coronary orifices; and as the aorta is decidedly the most frequent seat of chronic endo-arteritis, it was a fair inference that the peripheral vessels were healthy. The radials certainly were so, if this can be determined by palpation during life. Dr. H. asked if it were possible for such a degree of inflammatory thickening to exist without contraction. He had frequently observed, at autopsies of cases in which during life there had been no sign of cardiac disease, an opaque and thickened condition of the borders of the auriculo-ventricular valves, the valves being undoubtedly competent. Can this condition be referred to inflammation? If so, it must be acknowledged that inflammation in this situation is often harmless,—a doctrine which has heretofore found no advocate. It must be borne in mind by those who consider this change inflammatory, and explain the absence of contraction to want of time, that this is not a recent case. The symptoms were first complained of in December last, nearly a year ago, and disease may have been present long before. Dr. H. was inclined to think there had not been sufficient resistance in the arterial circulation to account for valvular changes.

Dr. Guitéras said that he desired to make a statement more fully explaining Dr. Fothergill's views. Dr. Fothergill lays particular stress upon

this increased vascular tension, in the early stages of the disease. He accepts the views of Johnson and Beale, who consider the thickening of the vascular walls at first as a muscular hypertrophy; that this is brought about by the contraction of the arterioles to resist the passage of a blood loaded with effete matters from disturbed assimilation and elimination. In this manner he explains the increased arterial pressure, which is certainly demonstrable by means of the sphygmograph.

October 25th, 1877.

Report of the Committee on Morbid Growths:

"A microscopic examination of the thickened mitral valve of the heart, presented by Dr. Henry, shows the thickening to be due to an increase in the growth of fibrillar connective tissue, in which are seen numerous nuclei. The endothelial covering, at different points, is seen to have undergone an increase in cellular elements. There was not observed either fatty infiltration or metamorphosis. Your committee are inclined to consider the thickening as a result of a slow inflammatory process."

November 8th, 1877.

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2. *A case of aneurism of the thoracic aorta, with unusual physical signs.*

Presented by Dr. William Pepper.

S., æt. about 38 years, had followed the sea from the age of 16 years. He was admitted to the Pennsylvania Hospital July 28th, 1877, suffering with symptoms of bronchitis, which were soon relieved, and he was discharged. He was admitted to the University Hospital on November 12th, 1877. He stated that he had enjoyed very good health until July last; and that since his discharge from the Pennsylvania Hospital he had been able to continue work as a sailor, and to make several voyages. Soon before his admission to the University Hospital he began to complain of cough, hard and dry in character, occasional shortness of breath, and pain in the region of the præcordia. These facts were not elicited until after the appearance of more serious symptoms.

From the 12th to the 17th of November he was about the wards, and seemed not to have much the matter with him. He was a well-built, stout man, of sallow complexion; and, as he had been recently in malarial districts, and as there was slight irregular febrile action, he was ordered full doses of quinine, and careful thermometric observations were made to determine the character of the febrile movement. On the 17th, how-

ever, he called attention to the increasing shortness of breath, and a careful examination of the thorax was made for the first time.

There was impaired respiratory movement of the left chest. On the right side the movement was marked; resonance was normal and the respiratory murmur was exaggerated. At the extreme right apex there were mucous râles over a very small area, and expiration over the same area was blowing and prolonged. On the left side there was absolute flatness on percussion in front, from the clavicle down to the third interspace where it was continuous with the cardiac dulness; laterally, there was vesiculo-tympanitic resonance from the anterior to the posterior borders of the axilla; posteriorly, there was marked dulness over the scapular region, while inferiorly, there was resonance but slightly impaired. These relations were not influenced by alterations of position of the patient's body. Inspection showed a distended vein which came from the left axillary region, crossing the pectoral region to reach the mammary vein.

On auscultation, over the area of flatness anteriorly there was entire absence of respiratory sound, with almost complete absence of vocal resonance and fremitus; posteriorly over the upper lobe there was feeble blowing breathing; laterally there was rather feeble vesicular murmur, and postero-inferiorly there was feeble vesicular murmur with scattered crackling râles. There was no aneurismal murmur heard over the area of dulness, nor posteriorly to the left of the vertebral column.

The area of cardiac dulness was increased, and the apex-beat was in the fifth interspace, one inch outside of the line of the left nipple. There was neither thrill, friction-sound, nor murmur, the sounds being merely distant and feeble. There was reduplication of the second sound, as heard over the body of the heart.

During the next two days the patient's condition was unchanged. On the 20th, after a quiet night, he had a slight attack of hæmoptysis, which ceased soon after large doses of gallic acid had been given. The countenance now expressed anxiety, and the alæ of the nose moved strongly in respiration. Inspiration was especially labored. The pulse was 96. The temperature rose during the day to 100° F. The following day there was a slight hæmoptysis early in the morning, and the symptoms were all aggravated. On the morning of the 22d, after a spell of coughing, there was another attack of hæmoptysis. He did not complain of pain, but of general soreness over the left chest. The dyspnœa was greatly increased, and the pulse was more frequent, and weaker. The temperature rose to 103° F. Cough was frequent and smothered, with bloody sputa. The dulness on the left side had now extended over the whole pulmonary area;

and over the lateral and postero-inferior parts of this side there was obscure bronchial breathing with some coarse crackling râles. For the first time there was an indistinct sense of pulsation in the second left interspace, about an inch from the sternum. On the morning of the 23d there was still another attack of hæmoptysis, of greater quantity than before. The pulse was rapid and small. The temperature rose to 104½° F. There was orthopnœa, which steadily increased with continued hæmoptysis, and death occurred on the morning of November 24th.

The treatment consisted in the free administration of opium, digitalis, and quinia, with gallic acid and ergot to arrest the hemorrhage.

The *post-mortem* examination was made twenty-four hours after death. The sternum was removed, and the thoracic organs were examined first *in situ*. The pericardial sac was decidedly enlarged. The right lung was somewhat emphysematous, chiefly along the anterior margin; the upper lobe was partially collapsed. The upper lobe of the left lung came forward as far as during moderate inspiration, while the lower lobe was somewhat pushed backwards by the distended pericardium. On cutting open the pericardium there were thick shaggy layers of lymph over both surfaces of the pericardium, with about half a pint of turbid serum in the pericardial sac. The heart was moderately enlarged, its apex corresponding to the point where impulse was noted during life.

The right lung presented at its apex a cluster (three or four) of round cheesy nodules, encapsuled by fibrous tissue, and only softened in points; otherwise its tissue was healthy.

The upper lobe of the left lung presented a curious combination of several large peripheral hemorrhagic infarctions, with complete collapse, of long standing, of the remainder of its tissue. These latter parts were tough and leathery. The posterior part of the upper lobe was not so completely collapsed as the anterior.

The lower lobe presented externally a mottled appearance, patches of purplish black being thickly scattered over the surface. On section it seemed uniformly consolidated. Any portion of it sank immediately in water. The tissue, as seen in section, was red, resembling closely a blood-clot; it was too friable, and in places was slightly granular.

On raising the upper lobe of the left lung, and carefully tracing upwards the arch of the aorta, it was found normal till after the great vessels had been given off. At the beginning of the descending thoracic aorta it expanded into an irregularly spherical tumor, which had hollowed out for itself a space in the tissues of the central part of the left lung. This aneurismal sac, which was about two and a half inches in diameter, pressed

on the trachea to some extent, but it had pressed so strongly on the left primary bronchus as to have caused gangrenous ulceration, which had finally entirely severed the bronchus. At this point of pressure the aneurismal sac had given way, so that the blood had escaped partly into the left bronchus, which was filled with soft clots, and partly into the tissue of the root of the lung. There was only a slight degree of atheroma of the aorta either above or below the seat of the aneurism. The œsophagus was compressed in a slight degree by the aneurismal tumor.* There were no morbid changes found in any of the abdominal organs.

Remarks.—A careful review of the arguments for and against the existence of circumscribed pleural effusion, of consolidation of the upper lobe of the lung, or of intrathoracic tumor, led to the conclusion that the physical signs were due to collapse of the upper lobe of the left lung from occlusion of its main bronchus by the pressure of an intrathoracic tumor. The existence of a hydatid cyst being further excluded, it seemed probable that the tumor was aneurismal rather than solid in character. Had it been of this latter character, the existence of localized disease at the right apex would have rendered its cancerous nature probable. But there was no external primary growth; there was no cachexia nor emaciation; the age of the patient was opposed to such a view; and, finally, the repeated copious hemorrhages and the rapid development of the pulmonary lesions were also opposed to it. The indistinct pulsation in the second left intercostal space, which appeared only on the day before death, might have been expected on either supposition, as a solid growth lying in contact with the arch of the aorta might readily have a pulsation communicated to its mass. The diagnosis that was formulated during life was, therefore, that there was an aneurism of the descending thoracic aorta, entirely covered by the upper lobe of the left lung; that it had occluded by its pressure the bronchus leading to the upper lobe of that lung; that an ulcerated or gangrenous opening was forming between the aneurism and the bronchus; that there were small cheesy formations at the right apex. It will be seen that the post-mortem examination confirmed this view; and incidentally it has been shown how this condition accounted for the very unusual symptoms and physical signs that were present.

The existence of pericarditis was suspected during life; but the absence of friction-sounds, the position of the apex-beat, and the impossibility of

* The wife of the patient came to the hospital for the first time on the day after his death. She then stated that for some time there had been more or less difficulty in swallowing solid food, and also some hoarseness of voice. These symptoms were not mentioned by the patient himself during the time he was under observation.

determining satisfactorily the boundaries of cardiac dulness, made it difficult to decide upon. I would ask attention to the reduplication of the second sound, which is a more frequent sign of pericardial exudation and adhesions than is usually recognized.

There is nothing positive known as to the cause. The patient's occupation, the absence of any history of injury, the old cheesy nodules at the right apex, the patches of endarteritis on the aorta, all confirm the suspicion that it may have been connected with constitutional syphilis.

The case also presents considerable interest from a pathologico-anatomical point of view. The position of the aneurism, flattening out the upper lobe and entirely concealed by it, the grangrenous destruction of the bronchial tube, and the combination of complete collapse of the upper lobe of the lung with several large hemorrhagic infarctions, may be specially noted. But still more interesting was the condition of the lower lobe, which presented complete consolidation, due to coagulation of blood in its vesicles and minute bronchioles. This lesion was marked during life by such rapidly developing physical signs, and there were at the same time such marked febrile symptoms and progressive rise in temperature, instead of the more usual symptoms of extensive hemorrhage, that the comparatively small amount of pneumonia is specially instructive.

December 13*th*, 1877.

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3. *Cancerous heart (?) with dilated right ventricle; sudden death, with symptoms of angina pectoris.*

By Dr. T. D. Ingram.

The specimen I have to present this evening I believe to be one of cancer of the heart. It is from a patient dying suddenly with symptoms of angina pectoris,—Charles K., a German, aged 32 years, a spice-grinder, and a man of temperate habits. Though having a rather irritable disposition naturally, this I believe was possibly increased, as I learned that the man complained of pain in the chest, over the region of the heart. His wife told me that for a long time he could sleep with comfort only while lying upon the right side.

The man frequently suffered from a slight cough, which he attributed to the dust from the mill, which was at times very irritating. He was regarded, however, by his fellow-workmen as somewhat of an athlete, being of a strong, muscular build.

In the early part of November I was called to see the man, thought to be dying in a paroxysm. I found him in bed, slightly propped up with pillows, and breathing with exceeding rapidity. There was no apparent dyspnœa or difficulty in the act, except that it was of a short, shallow, quick or panting character, too rapid to be counted. There was no lividity of the face: indeed, it was rather pale; and the hands were also quite cool.

His expression was one of great anxiety, and when spoken to, or questioned, he answered hurriedly and in monosyllables, in keeping with his manner of breathing. In this he quite persisted; but when told to breathe slowly he could do so without difficulty for a while, but would again soon relapse into the same shallow or panting breaths.

The pulsations at the wrist were very weak and rapid. On examination of the heart, I found its action irregular, exceedingly rapid, and feeble. An examination showed the pupils to be quite sensitive to light; and indeed, as the mind was entirely clear, and the breathing not absolutely embarrassed, any serious disturbance of the prominent vital functions seemed limited to the heart.

His wife told me they had retired as usual, and that she was awakened by his peculiar panting respirations. He then complained of intense pain in his breast and left side,—a sense of suffocation, or rather that an increase of the pain would ensue with a deeper breath. There seemed, indeed, to have been no exciting cause. Thus I found him at about 2 o'clock in the morning, and, as he complained of excessive pain, at once I injected a full quarter-grain of morphia under the skin of the arm. I requested him to breathe more slowly, as I deemed that his symptoms were being aggravated by the shallow respirations. I then found that the breathing gradually became slower and deeper.

The heart's action was still exceedingly weak, and I sent for tinct. digitalis; of this I gave at once a half-teaspoonful dose, and repeated it in less than an hour, as I found the pulse to be gaining in strength and becoming slower. After giving the second dose, the strength of the pulse improved greatly, and the respiration became much more natural.

Leaving directions to continue the digitalis, ten drops every two hours, and a quarter of a grain of morphia to be given by the mouth if the patient did not sleep, I returned home, having spent about two hours with the man, promising to return again at 5.30 A.M. When I called at the house in the morning, I found it closed; and, as the lights were out, I presumed my patient was asleep, and left.

Being called out of town, I did not see him until the following day, when I found him about and enjoying his usual health. On examining

the heart, I could discover no murmur, nor any irregularity, except a somewhat excited and peculiarly muffled action.

I now prescribed a large belladonna plaster, 6 by 6, and bade him wear it continuously, and tinct. digitalis ten drops three times a day. He called on me a week afterwards, complaining of a cough, which he referred, as before, to the irritating dust of his employment. I again examined the heart, and, as before, found no decided murmur, but, as I well remember, a peculiar muffled quality of its sounds.

I was next called hurriedly to see him on the evening of the 27th of November, three weeks after the former attack, the messenger saying he was dying in a spell. When I reached him, he was indeed dead, sitting in a chair, with his head and arms hanging over the back, presenting a most ghastly picture. He had been at his usual work during the day, but complained in the evening of pain in his chest. Rather later than usual, however, he ate quite a hearty meal. Later in the evening he was engaged with some friends playing at cards. At the end of the game he asked for something to drink, as he was thirsty and felt the pain again in his chest; but, before the drink could be handed to him, he had got up, and was staggering, saying that everything looked black to him. The attendants assisted him to an outer room and placed him in a chair, whence he had been moved to another chair within the house, all occupying less than five minutes, when I found him dead.

The *post-mortem* was made thirty-six hours after death. We found the left lung adherent throughout to the chest-wall and diaphragm; the right lung was adherent above, but not to the diaphragm; it was slightly adherent to the pericardium.

The lungs were darkly congested posteriorly, less marked in front. They contained much dark frothy blood, but were everywhere crepitant, with no evidence of solidification or of other masses.

Within the pericardium there was about an ounce of yellowish and slightly-turbid fluid. The pericardium was nowhere roughened or adherent to the heart.

The heart was evidently enlarged, more especially in its width, by the right ventricle having a full rounded border. Closer examination showed the under or posterior surface of this enlarged ventricle, near the apex, to be deeply injected, or of a sort of mottled redness. When felt, it offered a marked resistance, or the sense of some infiltrated substance giving it much greater firmness than the remaining tissue.

On opening the right cavities we found several tuberous projections,—the largest about the size of an ordinary chestnut,—occupying a position

on the ventricular septum near the auriculo-ventricular orifice, and binding down the smaller leaflet of the tricuspid valve. Other, though somewhat smaller, projections were scattered throughout the ventricular cavity, springing from both the septum and the outer ventricular wall. They were seated, apparently, immediately beneath the endocardial membrane. Much more marked, however, were the growths near the apex,—where they appear on the inside as elevated masses, and besides seem to be infiltrated throughout the muscular structure, reaching the outer or pericardial surface, as before described.

The cavity of the ventricle was markedly dilated, and its remaining walls thinned. The valvular leaflets at both orifices were otherwise healthy, though possibly insufficient from the dilated condition of the ventricle and auricle, as well as of the root of the pulmonary artery.

On opening the left ventricle the muscular wall seemed somewhat softened, and its cavity to be slightly dilated. The valves at both orifices were healthy and quite sufficient. Microscopical examination of the muscular fibres of the left ventricle showed very plainly the normal transverse markings, though in some fibres there were scattered small granulations, indicative of slight degeneration.

No careful examination of the abdominal viscera was made. The liver and stomach were observed in place, and were apparently normal. As no symptoms referring to them were observed during life, and not supposing cancer, we neglected to examine them, as well as other points more frequently the seat of such primary diseases.

I may add, here, that no history of hereditary cancer could be obtained, nor is there any evidence of a possible syphilitic taint, to which these new growths might be referred.

I may be permitted to add the symptoms of another case of angina pectoris, which I have seen since the one just described, in a man presenting himself at the Jefferson College clinic. This man, also only 32 years of age, complains of paroxysmal attacks of pain in his chest, about the region of the heart, and extending to either side. He also feels the pain in his arms and hands, where he frequently has it relieved by rubbing. Active rubbing, he says, will also relieve the pain in his chest.

In this case, I found no difficulty in discovering a quite sufficient lesion of the heart. For, upon examination there is very evident enlargement of the organ, the apex reaching a point considerably without the left nipple, and upon auscultation there is a short systolic aortic murmur, followed by a loud harsh murmur at the same orifice.

The heart, although apparently beating strongly, gives but a feeble as

well as a receding pulse. From the lesion of the valves, there is aortic regurgitation, and consequent dilatation of the left ventricle; this has not been followed by compensating hypertrophy.

To these notes I trust I may still be permitted to add some others, that I have quoted with reference to the history of cancer of the heart. In a short reference to the disease in an article in *Ziemssen's Cyclopædia of Medicine*, carcinoma is said to be the most frequent of the new growths of the heart, though a disease quite rare in itself. In 17,407 *autopsies* collected by two observers, cancer of the heart is said to have been found in only twelve cases. Again, in 4547 *autopsies*, of which 477 were cases of carcinomatous disease, cancer of the heart occurred nine times, and cancer of the pericardium seven times.

Cancer of the heart is much more frequently a secondary than a primary affection. Primary cancer of the heart may occur, and is generally of the colloid or melanotic form. Only one case of epithelial cancer of the heart is noted, and that by Paget.

I quote also from an article on the subject in *Reynolds's System of Medicine*, where it is stated that out of forty-five cases of cancer of the heart only *two* were definitely of a primary character, and in these the disease existed in only a part of the organ; though in seven other cases no mention was made of its existence elsewhere than in the heart.

Still quoting from Reynolds, "the most frequent location of the disease seems to be in the right ventricle or auricle, though it may occur in other parts."

The deposit may either assume the form of distinct tubera, of various sizes, seated either beneath the pericardium, or more rarely beneath the endocardium, or it may be found infiltrated within the tissue of the walls or septa,—the latter rare.

Still further from Reynolds I have noted that many observers have been led to believe that a majority of cases of angina pectoris were due to some obstruction or disease of the coronary arteries, with consequent faulty nourishment of the heart, leading to its dilatation. One observer found the statistics of this condition in twenty-one out of thirty-six fatal cases; another found that in twenty-four out of forty-five cases there was degeneration or disease of the aorta, reaching the valves or extending to the coronary arteries. In ten cases there was positive disease, and in twelve other cases there was preternatural softness of the tissues of the heart itself.

Many authors and observers, including Trousseau, have noted the symptoms of angina pectoris, in a more or less characteristic degree, asso-

ciated with thoracic aneurisms, especially those arising from the ascending part of the arch, near to or pressing on the heart itself.

In regard to the determination of how far any organic disease, and what special kind of organic disease, may have had to do with the symptoms of angina pectoris, I again quote from the article in Reynolds: "It is indeed at times very difficult; for, in the first place, organic disease may exist, without the possibility of its discovery, and, secondly, they are precisely the forms of organic disease most difficult of discovery that have been shown to be most frequently associated with deaths from angina pectoris."

The author says emphatically, though, that where the symptoms are less urgent, or in cases where the paroxysms become more frequently repeated, if the lesion can be distinctly made out by a careful examination, then the diagnosis becomes of the greatest importance, both to the physician and to the patient, in regard to prognosis and treatment during the intervals. *December 13th*, 1877.

Report of the Committee on Morbid Growths:

"A microscopic examination of the new formation developed in the walls of the heart presented by Dr. Ingram, demonstrates it to consist of lymphoid cells, spindle-shaped cells, and cells characterized by large granular round or oval nuclei, surrounded by a granular substance, suggesting very much the appearance of epithelial cells. There is a visible attempt to the formation of alveolar spaces by fibrillar connective tissue; but we also find between the cells a fibrillar intercellular substance. These elements, and their arrangement, give, in our opinion, the characteristics of a sarcoma,—variety alveolar. Muscular fibres in a state of granular degeneration, and undergoing inflammatory action, are seen in connection with the new formation." *January 10th*, 1878.

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4. *Case of stenosis of the aortic valves, with extensive calcareous degeneration reaching nearly to the mitral valve.*

By Dr. J. Solis Cohen.

The case from which the specimen was obtained was under my care at the German Hospital from October 27th, 1877, to December 7th, 1877. The patient was a medium-sized man, 50 years of age, of light complexion, and by occupation a regulator of gas-meters. He believed himself to have been healthy until about six months before admission, at which time he

was taken ill with malarial fever, followed by icterus, ascites, and great general debility.

His physical condition on admission was deplorable; icterus was very marked, ascites was considerable, with serious dyspnœa, anorexia was complete, and there were intense presystolic and post-systolic cardiac murmurs audible before the ear reached the chest. The diagnosis was probable cirrhosis of the liver, with stenosis of the aortic valves.

Under the external use of nitromuriatic acid, and the internal administration of nitromuriatic acid, digitalis, and jaborandi, the ascites subsided rapidly, the icterus diminished, and the appetite and digestion became good, and under a subsequent invigorating treatment with nux vomica and cinchona, improvement in the general health was very prompt, and the patient was discharged, with injunctions to resume work cautiously, and never to undertake more than half a day's work in the twenty-four hours, complete retirement from labor being out of his power.

Considering himself well, he resumed work at full hours, and one week later word was brought to the hospital that he had been found dead in bed that morning. A *post-mortem* examination of the body was made by the residents of the hospital, Drs. Berlet and Mellersh, who brought me the heart now exhibited, and reported that the body generally was in good condition, with plenty of adipose tissue, the liver was cirrhosed, the heart was stenosed at the aortic orifice with calcareous deposit, which extended beyond its limits; and the remaining organs appeared normal. I am indebted to Dr. Morris Longstreth for the following description of the specimen:

The heart is moderately enlarged, the enlargement being confined to the left side, the right ventricle appearing as a patch on the side of the organ.

The *right* auricle and ventricle, with the openings and valves on that side, were normal.

The *left* auricle was somewhat enlarged, its wall about normal, certainly not thickened; the endocardium appeared thickened somewhat.

The *mitral orifice* was found dilated considerably. The valvular leaflets are nearly normal, and likewise the tendons.

The *cavity of the left* ventricle is dilated, and the ventricular walls about normal or perhaps slightly thicker.

The chief disease is at the *aortic* orifice; this is narrowed to a slit-like orifice, allowing merely of a nickel piece being passed edgewise through it. The two anterior cusps were firmly united as far as their middle part along their line of mutual contact. The posterior cusp was still slightly flexible at its posterior part. There is a large calcareous deposit, extend-

ing not only throughout nearly the whole extent of the semilunar valves, but reaching down to the origin of the mitral leaflet.

The aortic sinuses are reduced in size; the aortic wall at this portion is nearly normal, except at a few points near the opening of the coronary arteries, where are seen small atheromatous changes. These arteries have their walls normal, and surrounded by a thick bed of fat.

The ascending part of the aorta is enlarged slightly, though the wall of the vessel is smooth and of normal texture. Atheromatous changes are visible where the great vessels leave the arch of the aorta.

Microscopic examination of tissue of left ventricle showed heart-fibre highly granular and fatty; the striation had largely disappeared.

Dr. Longstreth said that the specimen was a good illustration of one form of aortic valvular disease, with the consecutive changes resulting in the walls and cavity of the left ventricle.

The calcareous degeneration, with thickening and stiffening of the flaps, has narrowed the aortic orifice to such an extent, that it is a wonder any amount of hypertrophy of the muscular wall was able to propel the blood into the aortic system. From the advanced stage of the disease of this valve, the deviations from the normal in the left ventricular wall differ from those we find in simple moderate aortic stenosis, and approach those resulting from insufficiency of this orifice. In pure aortic stenosis, due, for example, to calcareous deposit limited to the peripheral part of the cusps, whilst their free borders remain flexible, hypertrophy of the wall of the left ventricle takes place, and this change is usually almost the only alteration in the form of the heart.

If, however, as in this specimen, stenosis becomes extreme, the final state is that the same disturbing force acts, or rather the same state of blood-distribution is conditioned, as in insufficiency of the valves, viz., the quantity of blood to be expelled by the contraction of the left ventricle is greater than normal. The mechanism of the condition in the two cases is very different. In insufficiency, the increased amount comes from a reflux of blood once expelled into the aorta; in stenosis, it is from blood unable to pass the narrowed opening. This condition is the terminal one resulting from the two forms of change at this valve. Let us see by what steps the condition is reached. In specimens where regurgitation is at death a well-marked phenomenon, the initial step in the morbid change is invariably an obstruction, however slight, to the exit of blood. The only exception to this rule is formed by examples of sudden rupture of a semilunar flap. The endocarditic process, with the subsequent vegetations on the leaflets, forms a sufficient resistance or stimulant to lead to over-action

of the heart-muscle,—the first step, therefore, in hypertrophy; later, if these obstructions grow greater, the hypertrophy increases; later still, the retraction of the flaps allows regurgitation, itself to be viewed as an obstruction to the onward flow of the blood. But regurgitation is now occurring into a hollow muscle, no longer of its ordinary normal thickness, but hypertrophied and accustomed by previous over-action to propel the blood with more than normal force. The small amount of regurgitated blood, at the beginning, serves, by the slight over-distention of the ventricle, to stimulate the strengthened muscle to still more forcible expulsive effort. The result is, therefore, hypertrophy of the wall, with temporary greater than normal expansion, at the height of diastole, of the cavity, but not permanent dilatation.

Dilatation, which invariably comes with insufficiency at this orifice, has its cause in the degeneration of the heart-fibre, dependent on two factors: 1, the over-action and the too frequent action lead to greater waste of nutrition than time allows for repair,—both alone insufficient to produce dilatation; 2, the lessened flow of blood in, and the increased heart-tissue to be nourished by, the coronary arteries, furnish the further needed cause in the process of decay. Degeneration of the heart-muscles means death; and, so soon as the amount of blood within the cavity rises in quantity above that which the muscular force is able to expel, the circulation must cease.

In stenosis, as in regurgitation, while the final condition, viz., degeneration and dilatation, is the same and has been arrived at by a like gradual process, the cause of accumulation of blood in the left ventricle is totally different.

The steps of the process are likewise very different: throughout the disease, when stenosis alone has been present, the continual tendency is to hypertrophy, dilatation occurring only in the last stages and after stenosis has become extreme. The gradual formation of the obstruction is as gradually equalled by compensatory hypertrophy: so long as the balance between opposition and muscular power is maintained, the cavity is emptied at each systole and no force is at work to produce dilatation. When the balance topples by the obstruction growing greater, degeneration allows of dilatation of the overworked and insufficiently nourished muscle. It is probable that this result comes sooner in insufficiency than in pure stenosis, for more reasons than one: in the first place, the ventricular cavity fills more quickly and stimulates the muscular fibre to a quicker-coming systole, so that in regurgitation the pulsations are more rapid than in pure aortic stenosis; the pause between the contractions—the time of repair—

is lessened; secondly, the coronary circulation is less well maintained under conditions of regurgitation, and is less perfect, in a rapidly contracting heart.

So long as the hypertrophied muscle continues equal to the demand made of it, the circulation is maintained at the normal in pure stenosis, and the patient presents no symptoms of the grave lesion, the physical signs alone making us acquainted with the condition; overwork, physical or mental, or other disturbing causes, however, are very apt to occasion temporary cardiac symptoms, which disappear with rest and quiet.

Death occurs in the marked cases of pure stenosis, as he believed was the case with this specimen, before the extremes of degeneration and dilatation result which we see in the organ from other forms of disease. The obstruction becomes so complete in a valve whose cusps remain rigid and expanded alike in diastole and systole, that the muscle, unable to move the blood through the slit-like opening, is paralyzed, and the paralysis comes suddenly; the heart stands still, and death is by syncope.

The clinical history of the case agrees with the common experience, that *aortic* valvular disease brings far greater danger of *sudden death* than morbid conditions at any other valve. This observation depends on the difference in construction of the flaps of the aortic orifice, as compared with the mitral leaflets, and on its position relative to the general circulation, as compared with all the other orifices. The ready retroversion of an aortic cusp or its rupture or laceration when diseased, followed by sudden emptying of the aorta and lowered blood-pressure in the aortic system and overdistention of the left ventricle, explains how slight is the bulwark holding back sudden death from syncope. It is not strictly accurate to trace death, when it come through such an accident at the aortic orifice, to syncope alone. True, the rupture of a flap empties, or so lowers the blood-pressure in the aortic system that cerebral anæmia and syncope occur. The symptoms in such cases prove it to us: the patient staggers and falls, like one shot. But one does not die from a single failure of systole, nor does death come instantly even from rupture of the aorta within the pericardium; both conditions tend equally either to lower blood-pressure or to empty the aortic system. In these two instances, the aortic valves being perfect, the succeeding systole restores blood-pressure, in the first case completely, in the second instance at least partially, so that fatal syncope is averted, and death comes slowly from hemorrhage. The true explanation lies in the condition of the left cavity itself. The sudden reflux of blood from the aorta, while the mitral orifice is delivering its normal quota, so stretches the ventricle that its unhypertrophied muscle stands still or inef-

fectually struggles with the newly-imposed burden; no blood, or very little, is expelled into the aortic system; after a few flutters the heart ceases action.

The specimen which Dr. Cohen has shown this evening is a marked contrast to one which he obtained at a recent *autopsy* at the Pennsylvania Hospital. The general alterations in the form of the heart, as well as the symptoms, were different; but especially did he wish to allude to the difference in the mode of death, although in the two specimens the disease at the aortic orifice was the same, viz., nearly complete obstruction without insufficiency. In the specimen to which he referred there was in addition mitral narrowing and rigidity (stenosis and insufficiency). From the coexistence of the valvular disease at two orifices, or rather from the presence of mitral disease, a difference in the form of the heart, in the two cases, was a necessity; the symptoms and the physical signs were marked by a preponderance so complete of those dependent on the mitral lesion that any one, seeing the case for the first time in its terminal stage, would have failed to detect the very marked aortic disease. There is also nothing unusual in this observation, which accords with the not infrequent experience in valvular lesions at these two orifices. But the chief contrast between the two consists in the respective modes of death: in one case, after a comparatively symptomless course, with only disturbances of the circulation resulting from overwork, a patient, who felt so well that in spite of warnings he continued his usual occupation, dies without any of the common premonitory symptoms of heart-disease; in the other case, the aortic disease commenced prior to that of the mitral, and, so far as is known of the patient's history, he was free from symptoms of disease, except temporarily after severe exertion, for a considerable period. Later, during the time he was under observation, the mitral lesion caused the most marked difficulties. The narrowed ariculo-ventricular orifice, with, at the same time, insufficiency of the valve, caused dilatation and hypertrophy of the left auricle, pulmonary congestion, and finally also general venous congestion. The backing up of blood into the right heart led to hypertrophy, and finally dilatation of each of its cavities, with dilatation of the tricuspid orifice. Death resulted from asphyxia, with its prolonged agony. It was a death from the right heart, not from the left, as in Dr. Cohen's specimen. *December 27th*, 1877.

5. *A case of heart-disease, followed by apoplexy.*

Presented by Dr. F. Turnbull.

J. L., æt. 38, laborer, admitted January 24th, 1878, to the Presbyterian Hospital. On admission, the following history was obtained. With the exception of slight rheumatism in the right shoulder, he had never been sick until three months ago, when he noticed that while lifting he became giddy and short of breath. He thought he had been obliged to micturate oftener than natural for several years. He also noticed that his feet swelled at night since he had felt unwell. Family history fair.

Examination.—Heart, region of cardiac dulness larger than normal. Impulse diffuse. A loud systolic cooing or musical sound; best heard to the outer side of the position of the apex. A less-marked systolic blowing sound, best heard at the second right intercostal space.

Pulse (radial) weak, and could not be felt when the arm was raised above his head.

Urine, not albuminous.

On the night of the 4th of March he called the night-nurse to him, and asked for something to move his bowels, complaining that he had a splitting headache. He was helped into bed by the nurse, who noticed that his legs seemed powerless. The nurse then came to me, saying that the man wanted a cathartic, and I ordered two comp. cath. pills. These were given him, and the nurse left the ward for a minute, but was called back to find the man lying in bed helpless and breathing in a peculiar manner. This was at midnight. I was immediately called, and, on seeing him, found that his breathing was stertorous, the pupil of his left eye dilated and that of his right eye contracted and sluggish. All his limbs except the left arm seemed paralyzed; but on pricking the soles of his feet slight movements of the toes took place.

His pulse was regular, pretty full, and about eighty a minute. His carotids were throbbing. His face was flushed and hot.

About eight ounces of blood were taken from his arm, with temporary relief. Mustard-plasters were applied to his feet, and two drops of croton oil placed on his tongue. At 2 o'clock A.M. the temperature in the right axilla was 99° F; in the left axilla, $88\frac{4}{5}$° F.

At 3 o'clock A.M., $102\frac{1}{2}$° F. in right axilla, and $101\frac{1}{2}$° F. in the left axilla.

He died at 3.40 A.M.

Post-mortem examination.—Heart.—Hypertrophy of left ventricle, its

walls being ¾ inch thick. The weight of the heart was one pound. The pulmonary valves were about normal. The aortic valves were replaced by vegetations constricting the orifice. The mitral and tricuspid valves were both thickened and contracted. No evidences of pericarditis found.

Lungs.—Old firm pleural and pleuro-pericardial adhesions of right side.

Brain.—On opening the dura mater, a large clot was found between it and the arachnoid, compressing the left hemisphere considerably. It covered the anterior and middle lobes entirely. A smaller clot, also superficial, was found over the anterior lobe of the right hemisphere. There was a small clot, separating the cerebral hemispheres, in the anterior half of the longitudinal fissure.

On examining the base, the clot of the left side was found to extend down in front of the medulla and backwards beneath and above the tentorium cerebelli, so that the two masses of the cerebellum were separated a little by it. The middle and posterior fissure of the skull on both sides contained some of the clot.

The origin of the hemorrhage was found to be in the situation of the left posterior cerebral artery, at about the middle of its course. The brain-substance was ruptured at this place. The wound was in the posterior lobe of the left hemisphere, and its opening at the base about two inches in diameter. The pia mater and arachnoid were torn through, and the blood had forced its way into the lateral ventricle of that side, filling it entirely. In the posterior corner of the right lateral ventricle was a small clot which extended anteriorly in diminished size; veins of the surface of the brain congested.

There was a lymphatic tumor of the neck just below the jaw, on the left side, which might have pressed on the jugular veins. It seemed, however, during life to be perfectly movable. It was not thought necessary, at the *post-mortem* examination, to dissect it. *March 14th*, 1878.

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6. *Specimens from a case in which death had resulted from meningeal hemorrhage.*

Presented by Dr. William Pepper.

I. W. P., æt. 76 years. Very strong constitution. Accustomed to drink large quantities of whisky and water daily. Began to fail in memory and mental power in 1876. Movements grew heavier and less powerful. Giddiness became marked in 1877, and continued with marked fluctuation till death. Hard of hearing, but no Ménière's disease. Occasional examination of urine at various times, up to within a few days of

death, showed no albumen. Pulse regular. Radials hard. Extreme arcus senilis. No cardiac murmur. Aortic 2d accentuated. In July, 1877, he began to have occasional sudden falls, with unconsciousness, but without convulsions. These falls occurred a dozen times at irregular times, and were apparently caused by extreme vertigo. He usually fell backwards, striking the back of the head. All the symptoms had been growing more aggravated, when on May 1st, 1878, he fell with unusual force, causing contusion of the scalp over the postero-inferior part of the left parietal bone. He was stunned for some time; then became restless, tossing the arms about, talking excitedly, and trying to leave the bed. This excitement was marked for two days, when it gradually became replaced by dulness; breathing grew puffing, and the pulse began to be slower, falling to 56. For the next four days stupor slowly deepened, with occasional spells of excitement, progressively growing less marked. The breathing gradually assumed a tidal character, which, on the fifth day after the fall, had become so pronounced that intervals between groups of respirations were thirty-seconds long, the respirations being hurried, in groups of about 15, beginning with a faint breath, rising to loud, noisy stertor, and falling again to gentle breath before ceasing entirely for one of the long intervals. The pulse continued to fall steadily in frequency, remaining regular in rhythm: it was successively 50, 40, 32, 26, 20, 16, and finally, during the fifth and sixth days after the fall, remained at 12 or 13. For twenty-four hours before death, which occurred on the seventh day, the respirations grew regular and more frequent,—32 in a minute,—while the pulse continued at 12 in a minute. Unconsciousness was profound. While tidal breathing lasted, the pulse-rate was very slightly influenced by the breathing: during the respiratory periods the pulse rose to the rate of about 13, and during the intervals fell to the rate of about 11. Urine was passed involuntarily, and was not albuminous. Power of swallowing was retained till thirty-six hours before death, which occurred from increasing pulmonary congestion and œdema.

Post-mortem.—Head.—Skull very dense; diploë completely indurated. Extensive meningeal hemorrhage over left hemisphere; the clot, which was dark and soft, was chiefly seated over the posterior lobe, and had gravitated to the base, where it pressed on the tentorium. Very little, if any, blood had actually entered the cerebellar fossæ. The blood had evidently escaped from one of the meningeal veins, which presented a perforation of its walls, partly filled by a clot. There was no hemorrhage into the ventricles, or into the substance of the brain. The arteries at the base were highly atheromatous.

Thorax.—Lungs healthy, but deeply congested. The heart was surrounded by a great quantity of fat. The muscular tissue was friable and pale. Calcareous nodules were found at the base of the mitral and aortic leaflets, but the valves were competent. The aorta presented only small patches of atheroma. The coronary arteries presented a most intense degree of calcareous degeneration, being converted into rigid and brittle tubes.

Abdomen.—Liver fatty and slightly cirrhotic, and also presented nutmeg congestion. Kidneys congested; cortex presented small cysts, and also a moderate degree of interstitial disease, with some contraction and granulation.

There was a remarkable amount of soft yellow fat over the omentum, and around the pelvic viscera. *May 9th*, 1878.

Report of the Committee on Morbid Growths:

"A microscopic examination of the specimens presented by Dr. Pepper.

"The kidney is found to have the intertubular connective tissue very much increased in amount: the capsules of the Malpighian bodies much thickened, also the connective-tissue wall of the blood-vessels. The cells within the uriniferous tubules are enlarged, and in an advanced stage of granular degeneration; many of the tubules are entirely devoid of cells. The pathological change is one of interstitial nephritis.

"The liver is found to be in a state of indurated hepatitis, shown by a very decided increase of the connective tissue, and the excessive granulation of the cells.

"The muscular tissue of the heart has in a great measure lost its transverse striation, and the fibres present the morbid appearance of fatty metamorphosis.

"The large arteries from the brain present numerous points of atheromatous change, which have undergone calcification. The capillaries of the substance of the brain are seen here and there in their course to have a granular deposit resembling a calcareous infiltration of their walls."

May 23d, 1878.

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7. *Three cases of heart disease associated with kidney disease.*

Presented by Dr. James Tyson.

George C., colored, æt. 45, native of Philadelphia, occupation barber, was admitted to the Philadelphia Hospital, April 5th, 1878, in a very feeble and prostrated condition, frequent pulse, general œdema, and great

dyspnœa. He was a very large, stout man. Although his intelligence was perfect, he never was inclined to answer questions. He said he had been healthy previous to his present illness, which had been of but a few weeks' duration; he denied having had rheumatism or syphilis, or having used liquors in excess. Bowels constipated; tongue coated; no appetite; great thirst. Cough with white frothy expectoration. Feet very much swollen; whole body œdematous. Skin dry. Pulse 120, dicrotic. Urine diminished in quantity, and highly colored.

Physical examination.—Area of heart's dulness much increased; systolic murmur at the base; later, also, a mitral systolic murmur was observed. There were moist râles all over the chest and back. Examination of urine revealed albumen in variable quantity at different times, from one-quarter of bulk examined, to a trace; no tube-casts, but often much epithelium from the kidneys, and a great excess of urates.

Diagnosis.—Aortic stenosis and slight mitral insufficiency. He improved for a short time under digitalis and tonics. A jaborandi sweat did not act favorably. Soon his condition grew worse; œdema became more marked; and the patient was compelled to remain in a sitting position during the last two weeks, on account of excessive dyspnœa. He died May 12th, 1878.

Autopsy.—*Heart* hypertrophied and dilated. Aortic valves slightly atheromatous and stiffened, also fenestrated, but not thickened. Mitral cusps evidently, but not markedly, thickened. Aorta dilated, and highly atheromatous.

Lungs œdematous; some pleuritic adhesion on the left side.

Kidneys enlarged and swollen; in a state of parenchymatous nephritis.

Liver and *spleen* normal.

Brain not examined.

Remarks.—The most interesting facts in the history of this case are the slight degree of valvular lesion compared with the extreme degree of the symptoms, the marked dyspnœa and excessive dropsy, and the fact that there was also chronic tubal disease of the kidneys, with very few casts.

J. M., colored, æt. 56, native of Virginia, was admitted to the Philadelphia Hospital four times. First admission in May, 1877, for one week, with the diagnosis of aortic regurgitation. Second admission in July, 1877, when he was transferred to the venereal wards. Third admission in December, 1877, when he remained in the medical wards until March last, under the care of Dr. Guitéras; diagnosis, chronic Bright's disease. Last admission, May 24th, 1878, under my own care. When I saw him

he was in a very feeble, prostrated condition, frequent pulse, great dyspnœa, and not able to speak.

Physical examination.—Dulness on percussion over the right side of chest; region of dulness changed by change of position of patient. The heart was pushed much to the left side. First sound of heart reduplicated; slight systolic murmur at the base, and also a mitral regurgitant murmur. Dr. Guitéras also found marked dulness over the upper part of sternum; right pulse larger than left.

Examination of urine.—Albumen only a trace; urates in excess; hyaline and some granular tube-casts.

Diagnosis.—Pleuritic effusion; hypertrophy of heart; aortic stenosis and mitral insufficiency; interstitial nephritis. Dr. Guitéras diagnosed, in addition to this, atheroma and dilatation of the aorta.

Died on June 9th.

The *post-mortem* notes read as follows:

Heart.—Hypertrophy without dilatation; walls of left ventricle one inch thick; posterior leaflets of mitral valves thickened, the chordæ tendinæ of left valve much thickened, pulling out the musculi papillaris, which were extremely hypertrophied and unusually hard. Semilunar aortic valves slightly thickened, and atheromatous. Aorta greatly thickened, and generally atheromatous, and *dilated;* about one inch from its commencement it became double.

Lungs.—Right lung much engorged and congested, the lower lobe completely consolidated, and entire hemorrhagic infarction. Extreme pleuritic effusion on right side.

Kidneys contracted, not more than three inches long; granular.

Liver, *intestines*, and *peritoneum* healthy.

Brain not examined.

Microscopic examination of kidneys confirms an advanced interstitial nephritis.

George J., white, æt. 35, native of Philadelphia, a laborer, admitted to the Philadelphia Hospital, April 17th, 1878. He was a large, strong man, has never been sick, except an attack of rheumatism, which he had in 1871, for a few weeks, and never since. He had syphilis, and has been also a drinking man. His sickness dates only from February last, when he took cold, being one day whilst perspiring very much exposed to snow and cold wind. Had cough, fever, shortness of breath, and pain in the region of the heart. A few days before his admission to the hospital his feet began to swell, the swelling extending later over the whole lower extremities and the scrotum. He did not complain of any pain, but had

very great dyspnœa. Appetite good; excessive thirst; quantity of urine diminished—only a trace of albumen; no tube-casts; some epithelial cells from the kidneys, and some blood-corpuscles.

Physical examination.—Area of heart's dulness increased. Marked systolic murmur at the base of heart, and a murmur is heard in the right carotid; a mitral murmur was also observed coincident with the impulse, and transmitted to the axilla. Vocal fremitus diminished at the left side of chest.

Diagnosis.—Mitral insufficiency and aortic stenosis, and chronic pleurisy.

The patient died suddenly and unexpectedly, May 12th.

Autopsy.—*Heart* much hypertrophied; cavities much thickened; chicken-fat clot in right auricle. Extensive vegetations on aortic sigmoids, with partial calcareous degeneration of the same. Marked thickening of mitral valve.

Lungs.—Hypostatic congestion of both lungs; pleuritic adhesions on left side.

Kidneys large, swollen; capsule strips off easily. Left kidney with marked hemorrhagic infarctions; in both kidneys minute hemorrhages over the surface. Cortex increased. Microscopic examination reveals a parenchymatous nephritis, with capillary congestion and extravasation of blood.

Dr. Guitéras said that at a previous meeting he had referred to several cases where he had succeeded in diagnosing slight dilatation of the aorta by means of respiratory percussion. In some of the cases the diagnoses have been since that time confirmed by *autopsies*. This had led him to attach much importance to the systematic use of this method of percussion for examination of the aortic region. He made it a rule never to examine this region without percussing at the end of a forced expiration. As the lungs collapse over the large blood-vessels, it is surprising to find how clearly the area of dulness of a dilated aorta becomes apparent.

The first case he had examined after this manner was one where aneurism had never been suspected, and where no dulness could be obtained by ordinary percussion, yet he was enabled to demonstrate to a large class an area of almost flatness over the right border of the sternum, above the third rib. Slight pulsation could also be seen towards the end of expiration. Other symptoms have developed since, leaving no doubt as to the diagnosis.

In Case No. II., a colored woman, 38 years old, the symptoms of pressure on the trachea were well marked. Symptoms of vascular disease rendered it probable that the source of pressure was an aneurism, but

the dulness at the upper piece of the sternum could only be detected at the end of forced expiration.

In Case No. III., an old woman, there were evidences of valvular disease, both mitral and aortic. The aortic systolic murmur was so feeble (weak ventricle) that the existence of lesion of the semilunar valves had been doubted. By respiratory percussion the presence of aortic dilatation was demonstrated, and the diagnosis was subsequently confirmed at the *post-mortem*.

Of Case No. IV. no notes had been kept. During life the enlargement of the vessel could only be demonstrated at the end of expiration. Diagnosis confirmed by the *autopsy*.

Case No. V. was the one now before the Society. When this case was admitted early this year in the wards of Dr. G. at the Philadelphia Hospital, he supposed it to be one of contracted kidney with cardiac hypertrophy. But his attention was called to examine the aortic region by a prominence of the right sterno-clavicular articulation. This had nothing to do with the aortic lesion, but pulsation at the base of the neck was noticed, with inequality of the radial pulses. Respiratory percussion led to the discovery of a well-marked area of dulness.

Case No. VI. was examined previous to these investigations, but it is mentioned because though the dilatation was not greater than in some of the cases just described, yet it could be detected by ordinary percussion. The emaciation of the patient probably accounts for this. She died of cancer of the uterus.

Case No. VII. was seen only yesterday, after a prolonged absence in Cuba. In 1876 Dr. G. had found evidences of disease of the aortic valves and of atheroma of the arteries in its early stage. During the same year he saw him, in consultation with Drs. Alonzo Clark and Austin Flint, of New York, and Dr. Landeta, of Havana. Dr. Flint confirmed the diagnosis, but Drs. Clark and Landeta suspected the existence of some dilatation of the arch. Dr. Clark based his diagnosis upon evidences obtained by ordinary percussion. Now the patient presents all the symptoms of aneurism of the ascending portion of the arch. It is very probable that respiratory percussion would have made the lesion more apparent in its incipiency. Curiously enough, at present, when there can scarcely be any doubt of the existence of an aneurism, the percussion resonance over the aortic region is but very slightly impaired, and is not influenced by respiration. This is due, Dr. G. believes, to adhesions fixing the lung.

He also desired to call attention to the value of pulsation of the neck, as being a very conspicuous symptom in cases of aortic disease, both of

the valves and vessel. It should always lead to a careful examination of the aortic region. In differences between the carotid pulses, and in unilateral thrill pretty strong evidence is found of disease of the aortic arch. Of seven cases presented, five had unequal radial pulses In No. II. this symptom was absent; in No. IV. it was not looked for.

Unfortunately, the specimen of double aorta was removed from the body before the anomaly was noticed. At the first glance it was difficult to determine whether the double aorta was not a dissecting aneurism. There was a slit about an inch beyond the left subclavian artery leading into the accessory aorta. The latter ran up to the subclavian, forming a pouch, and downwards as far as the mutilated specimen shows. Further examination leaves no doubt as to this point: the edges of the slit are smooth, the lining membrane of the anomalous channel has the normal appearance, and, most important of all, the orifices of the left intercostal arteries are seen in the outer wall of the false aorta; the right intercostals arise from the main vessel near the septum: in the latter some aborted orifices may be found.

As a point bearing on the pathogenesis of atheroma, it may be mentioned that, whilst the arch of the aorta is well dilated, and, together with the rest of the main channel, is highly atheromatous, yet in the accessory aorta, where the blood-pressure must have been much less, there is scarcely any atheroma. *June* 27*th*, 1878.

IV.—THE ORGANS OF RESPIRATION.

1. *Pneumonia of the apex, with unusual physical signs.*

By Dr. John Guitéras.

A. W., æt., 60; tailor; German; admitted to the Philadelphia Hospital on the 15th of November, 1877. Night-nurse did not think he was very sick. In the morning he was found at the window trying to get some air. When my resident, Dr. Musser, saw him, he was sitting by his table, leaning his head on it, and suffering with much dyspnœa. He was ordered to bed. He had fever, cough, no appetite, and the bowels were constipated. He had been sick about ten days, though at no time in bed. Has always been a very healthy man.

Inspection showed a marked prominence of the front of the right chest, especially towards the apex; *palpation*, a complete absence of vocal fremitus. At the level of the folds of the axilla the right chest measured two inches more than the left. Flatness upon *percussion* extended from the apex to the lower margin of the middle lobe of the lung in front, and was readily traced backwards and upwards along the side of the thorax in the direction of the line of separation from the lower lobe. On *auscultation*, the râles of œdema were found to be very abundant over the left lung and inferior lobe of the right. Over the area of dulness the same râles were heard, but appeared distant; the lung itself gave no sound; there was no bronchial breathing, no vocal resonance. A forced whispered expiration gave rise to a feeble and distant blowing sound, hidden somewhat by the tracheal gurgling, yet harsh and characteristic of consolidation. He died the next night.

Autopsy.—No emaciation or discoloration of the skin. The enlargement of the right upper segment of thorax is apparent to the eye. On removing the sternum the upper and middle lobes are found consolidated, and bulging out and towards the left about one inch beyond the median line. No adhesions of the left pleura. Recent adhesions of the right, and an effusion of about four ounces of serum, with shreds of lymph floating in it.

On removing the lungs, the two affected lobes were found to present

the same volume as the left lung and the lower lobe of the right; these latter portions were œdematous. The upper lobes presented the grayish marbled appearance of gray hepatization, exuding the characteristic reddish, purulent fluid. The tissue breaks down readily on pressure. In the middle lobe there are some spots of red hepatization. Except at the very root of the lung, the bronchial tubes are collapsed or filled with pus. Healthy portions of lungs weighed two pounds; the diseased, four and a half pounds. There was an *ante-mortem* clot in the right heart.

The other organs were healthy.

Remarks.—The lack of treatment, the age of the patient, the localization of the pneumonia, contributed to the fatal termination. Though pneumonia of the base is so much more frequent than pneumonia of the apex, yet I believe I have seen as many *post-mortem* specimens of the one as of the other.

The interest of the case is found in the physical signs. These clearly pointed to a circumscribed pleuritic effusion. I diagnosed pneumonia from the direction of the line of dulness alone. Sufficient attention is not paid to this very important element of diagnosis,—viz., the position of the inter-lobar lines. We all know how frequently they are boundary-lines for pathological processes.

The physical signs here were due to the absolute absence of air from the affected lung. The bronchial tubes were not patulous; even the larger ones were filled with thick inflammatory débris, thus interfering with the production of blowing breathing, increased vocal resonance and fremitus. The same factor entered with the pressure of the swollen lung against the chest-walls, in the production of absolute flatness.

November 22d, 1878.

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2. *Pneumonia with unusual physical signs.*

Presented by Dr. E. T. Bruen.

Jos. F., æt. 41, was admitted to Philadelphia Hospital December 7th, 1877. He stated that for two years previously he had suffered from catarrh in winter, but had never been obliged to relinquish his work in consequence of it. A week before his admission, he was taken sick suddenly with fever, with pain in the chest, and was obliged to go to bed. Two days after, he commenced to spit up some very offensive purulent matter.

Upon examination of his chest, dulness on percussion was found to

exist over the entire right lung posteriorly, excepting over an area of about four inches below the spine of the scapula, and between the scapula and the vertebral spines. At this point the percussion was very tubular, even tympanitic, in character. Anteriorly there was dulness from the clavicle to the second rib, but below this the percussion was very tympanitic. Laterally there was dulness. The patient complained very much of soreness over the region of the tympanitic resonance. The respiratory murmur, though everywhere feeble, was bronchial, excepting over the area of tympanitic resonance, where it became very hollow, cavernous. Below the spine of the scapula, posteriorly there was well-marked pectoriloquy, elsewhere the resonance of the voice was diminished. The left lung was filled with moist râles, but otherwise nothing abnormal was noted. Temperature was 102° F., pulse 120, the patient much exhausted by exposure and want of food. Expectoration was profuse, especially in the morning and evening; at other times there was but little, and the odor was very offensive, so much so as to render the air around the bed disagreeable. There had been but little loss of flesh.

A diagnosis of pneumonia was made, but the existence of a vomica in the parenchyma of the lung, or else bronchial dilatation, was strongly suspected.

The patient died suddenly the next day (December 8th). A *post-mortem* examination was made twenty-four hours after death. The right side of the heart was found almost filled with *ante-mortem* clot. Its structure was healthy.

The right lung was in the stage of red hepatization, passing into gray hepatization. No cavity or bronchial dilatation was to be found. There was a small pleuritic effusion at base of right pleural cavity. The left lung was congested, but otherwise normal. Examination of other organs revealed nothing unusual.

I would call attention to the physical signs present, because I have from time to time seen similar cases. The explanation given by some is, that when consolidation is very complete, we obtain tympanitic resonance from the vibration of air in the bronchial tubes.

In this case, the history of previous bronchitis, the location of tympanitic percussion, and the offensive character of the sputa, made me suspect bronchial dilatation, or a vomica. The history of these cases and the general symptoms are probably our safest guides to the correct interpretation of the physical signs. *December 13th*, 1878.

3. *Case of circumscribed epithelioma of the larynx.*

Presented by Dr. J. Solis Cohen.

The specimen is from the body of an unmarried English lady, aged about 50 years, of a long-lived family (both parents still living, over eighty years of age, and engaged in active and laborious business), who began to be troubled with a cough and occasional difficulty in deglutition about fifteen months before death (December 8th, 1877). The lady was treated for a while for bronchitis, and on placing herself under the care of her family physician, Dr. Macpherson, about December, 1876, that gentleman on a laryngoscopic examination detected an enlargement of the epiglottis, apparently œdematous. Little change took place in the condition of the patient until February of the present year, when she was garroted by a burglar whom she discovered in her bed-chamber, after which her symptoms rapidly increased in severity, the cough becoming more and more troublesome, and deglutition getting so difficult, that a proper amount of nourishment could not be swallowed. By May 3d, at which date I first saw her in consultation with Dr. Macpherson, the impediment to swallowing was so great, that we determined to commence at once with the systematic exhibition of nutritive enemata, and from that time forward, nutrition was carried on in that manner almost exclusively, there being but rare intervals at which swallowing was at all practicable, and the amount ingested being too insignificant to supplement the enemata in the least. Laryngoscopic inspection, at the date just mentioned, revealed a fungous-looking mass involving the entire free portion of the epiglottis and the left aryteno-epiglottic fold. There was considerable enlargement on the left side of the neck, greatest and hardest in the region of the carotid glands. The diagnosis was epithelioma, and this was confirmed by microscopic examination of several fragments, which were removed for the purpose, some of which were mounted by Dr. Seiler. I removed the entire free portion of the epiglottis, but made no attempt to remove the mass from the aryteno-epiglottic fold, as it was impracticable to determine how far the disease extended, although the character of voice was such as to indicate integrity of the vocal cords. After removal of the epiglottis the patient was able to swallow occasionally for some weeks. She was placed upon arsenic, quinine and brandy being administered per rectum in the nutritive enemas.

The remaining growth was treated by astringents, applied locally by Dr. Macpherson, to restrain hemorrhage and constringe the vessels. The

difficulty of swallowing soon increased to absolute aphagia: the patient became gradually weaker, and from time to time expectorated detritus from the mass. Towards the close of July, dyspnœa became superadded to the symptoms, and this increased to such an extent that I was suddenly summoned, late in the evening of August 9th, to perform tracheotomy, which I did without anæsthesia, with the assistance of Dr. Macpherson and my neighbors Drs. Mears and Turnbull, Jr., who kindly accompanied me to the residence of the patient for the purpose. The immediate effect of the operation was, the dyspnœa ceased, and, as I am informed by Dr. Macpherson, ceased for good. The patient made a rapid recovery from the operation, the wound healing with unusual promptitude, despite some local gangrene of the fistulous tract, which for a few days blackened the surface of the silver tube introduced. Swallowing, however, was not improved, and everything attempted to be taken into the stomach passed through the tube, owing to a cause which will be apparent on examining the specimen. The improvement immediately succeeding the operation was soon followed, as I am informed, by gradual progressive emaciation, with frequent discharge of fetid débris from the larynx, both by mouth and by tube; and the patient died exhausted on the evening of December 8th, 1877.

At mid-day on December 10th, Dr. Morris Longstreth made a *post-mortem* examination in the presence of Dr. Mears, Dr. Macpherson, and myself, and removed the parts exhibited to the Society, comprising the larynx, a portion of the trachea including the wound of tracheotomy with its overlying integument, the posterior portion of the tongue, portions of the palatine arches, pharynx, and œsophagus respectively, and two enlarged and degenerated glands from the left carotid region. The connective tissues were quite dense. The destruction is most marked on the left side of the larynx. The whole of the aryteno-epiglottic fold and its constituents down to the lower border forming the ventricular band (false vocal cord) of that side is destroyed, exposing the sac of Hilton from above, and leaving the ventricle and ventricular band intact. It is through this route that food gained access to the larynx on swallowing. The cartilages and vocal cords upon both sides are intact. The stump of epiglottis (chiefly the petiolus), which is the only portion of that structure remaining, appears not to have been involved in the morbid growth, but there are tracks of ulceration extending completely around it. The remaining portion of the larynx, the tongue, palatine arches, pharynx, and œsophagus, appear to be free from disease.

There were two lymphatic glands lying in the line of the carotid artery

of the left side, at the level of the larynx, which were enlarged. On section they showed cheesy degeneration in nearly their whole extent. The microscopic examination made by Dr. Longstreth showed abundance of oil drops, lymphatic cells, highly granulated and fatty, and also some crystals of cholesterine. There were also seen numerous large flat cells, containing a single large granular nucleus. These resembled pavement epithelium, but were, however, of greater thickness. The tissue surrounding the glands was very firm, dense, and white in color. Under the microscope it showed fibrous tissue with numerous small nuclei.

There was no microscopic evidence of an extension of the malignant disease to the glands or to the surrounding tissue.

The limitation of the disease to the larynx stamps it as primary without doubt; the removal by ulceration of all the abnormal growth stamps it as circumscribed. *December 27th*, 1877.

Report of the Committee on Morbid Growths:

"The larynx presented by Dr. Cohen, and referred to the Committee on Morbid Growths, shows from a microscopic examination of a thin section, taken from the remaining portion of the epiglottis, pegs formed of epithelial cells penetrating into the submucous tissue; here and there some of the cells forming the pegs are arranged concentrically, constituting the so-called pearly bodies. We therefore find an infiltration of the submucous tissue by an epitheliomatous growth." *January 10th*, 1878.

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4. *Gangrene of the lung following senile pneumonia.*

Presented by Dr. R. G. Curtin.

Mrs. W., aged 77, had a cough for four months, which was rather worse at night. The expectoration at first was scanty, but the second month she commenced to expectorate a tough, gelatinous mucus. About four weeks before her death her respiration became embarrassed, and the cough grew worse, accompanied by considerable fever; her pulse ran as high as 120 at times; some dulness posteriorly on the right side; coarse crepitant râles over the same region. Four days before death she began to expectorate a dark-colored and very offensive material. The cough was very harassing. She died slowly and quietly. This case was under the care of Dr. De F. Willard.

Post-mortem.—All the superficial arteries were very much degenerated.

The radial arteries were like a string of beads. The aorta on the inner surface was almost brittle.

Lungs.—Around the root of the right lung were found several pulpified spots, oval in shape, the largest being about two inches by one and a quarter inches. The lung-tissue in these places was simply melted down, being almost natural in color. The softened place was not surrounded by indurations, nor was there any evidence of congestive pneumonia, consolidation, or infarction. The odor, at the examination, was characteristic of gangrene of the lung. The bronchial glands were not enlarged, nor was there any evidence of cancer. *February* 14*th*, 1878.

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5. *Gangrene of the lung following latent pneumonia.*

Presented by Dr. R. G. Curtin.

A. B., aged 34, single. Previous to her last illness her health had been quite good. There was no hereditary tendency to tuberculosis. She had been sick with latent pneumonia about four or five weeks, when she began to expectorate material characteristic of gangrene of the lung. About three weeks after the advent of the gangrene she died of exhaustion.

Post-mortem.—The right lung was found to be diseased throughout. The middle and lower lobes were in a state of gray hepatization. The upper lobe was entirely destroyed by the gangrenous process, the cavity being traversed by the larger blood-vessels and bronchi, which had resisted the action of the disease. The other organs were healthy.

February 14*th*, 1878.

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6. *Fibroid phthisis and bronchiectasis.*

Presented by Dr. John Guitéras.

From the notes of my resident physician, Dr. Musser, I extract the following history. Susan M., æt. 56, single, domestic. Admitted to the Philadelphia Hospital November 17th, 1877. Family history very favorable. With the exception of typhoid fever, she does not remember having any acute sickness. For the past twenty-two years she has had a cough, with abundant muco-purulent expectoration; but she has been able to do general house-work up to four days before admission, although

she has been of late losing flesh and strength. She thinks the fever commenced a few days before admission. She never had syphilis.

On November 20th, I found her very weak and emaciated. My notes of the physical signs were as follows. *Inspection.*—Marked depression of the infraclavicular regions, more so of the right. Right nipple higher than the left. There is no retraction of the sides. Posteriorly, there is no difference in the shape of the two sides. There is decided deviation of the trachea to the right side. *Palpation.*—The left apex expands more than the right. There is no difference at the bases. The vocal fremitus is increased throughout the right side. Mensuration shows the circumference of the right side to be half an inch less than the left.

The only pulsation visible about the chest is at the second interspace, one inch to the right of sternum. There is also some pulsation in the epigastrium. A distinct impulse is felt at the former place, and a very slight one behind the cartilages of the left false ribs.

Percussion.—There is resonance throughout the left chest, encroaching somewhat beyond the median line in front, and replacing the cardiac dulness. The hepatic dulness begins at the fifth rib, and just within the line of right nipple it becomes continuous with the cardiac dulness. The latter extends up to the third rib, where the tympanitic resonance of a cavity commences.

Auscultation.—The respiratory movements are irregular. There is harsh breathing over the left apex. At the right apex there is marked cavernous breathing, and amphoric whisper. The gurgles and the heart-sounds, which are loudest at the second right interspace, do not present any metallic echo. The heart-sounds are inaudible at the left axilla, and well-marked in the right. At a previous examination I had found that the gurgles and cavernous puff were influenced by the cardiac rhythm during inspiration. Bronchial breathing is heard below the cavity, and the cavernous breathing is well marked behind.

Dyspeptic symptoms were well marked. No albuminuria. She had menstruated up to her forty-eighth year. The liver was slightly enlarged. The expectoration was muco-purulent, and of reddish-brown color. The breath extremely fetid. There had been no hæmoptysis. The temperature ran at first between 99° F. and 100° F., during the last two weeks it frequently reached 101° F. in the evening, and she had night-sweats. The pulse and respirations averaged 100 and 28. During the last week they rose to 120 and 40.

She lay in a listless condition, without any pain or dyspnœa, and finally died on the 21st of December.

Post-mortem.—The right pleura is adherent throughout, the left only at the apex. Except over the cavity in the right apex it is not very thick. The cartilages of the bronchial tubes are softened in some places, and they are distinctly seen through the thin mucous membrane. At some places the lung-tissue may be seen through the membrane. The transverse muscular fibres are also very prominent and isolated, as well as the longitudinal fibrous bands. There is no ulceration, except where the cavities have formed. The inferior right bronchus is dilated. The superior one is thickened, and about an inch from its orifice it merges into an enormous cavity. This occupies the superior lobe and upper portion of the middle. The walls of the cavity present many anfractuosities and bands of fibrous tissue. Its upper wall is made up by the pleura. The pulmonary tissue below the cavity is much indurated, and of a slate color. The section is extremely granular, and at some spots marbled with a grayish color. The mouths of the blood-vessels and bronchioles appear open on the surface of section. The lower part of the lung is less affected, but the bronchial tubes are thinned out, and uniformly though slightly dilated. Caseous masses are quite rare; they present a grayish color, they are very granular on section, and are broken down with great difficulty. They are evidently developing in young formations of fibrous tissue. The left lung is not so much affected, but the upper lobe presents some caseous masses of the more common appearance. The upper left bronchus presents a well-marked dilatation before it merges into a cavity much smaller than that upon the right side.

The right side of the heart was hypertrophied and dilated. The other organs were healthy. There was very slight lateral curvature of the spine.

Remarks.—It may be observed that though during life the position of the heart was so carefully determined, and found to be very unusual, yet very little mention of it is made in the *post-mortem* notes. The reason of this is that this *autopsy* was made in the usual manner, and was therefore worthless as far as studying the position of heart was concerned. I have more than once been surprised at finding a heart far nearer its normal position than the physical signs had led me to suppose. Even the auricles are not found in position to explain the pulsation detected perhaps at the second right interspace. The reason is obvious: on opening the thorax, the less affected and vicariously distended lung falls to near its usual place, and the heart, without support, also returns towards its normal position. I am sure that many *autopsies* are rendered worthless by our forgetting the differences that must exist between the distended lung, and the lung collapsed after opening the thorax. The relations are altered,

and I feel certain that physical diagnosis loses in this manner much of what it might gain from the *post-mortem* table. All this can be avoided by ligating the trachea before opening the thorax. A great deal may also be learned by inflating the lung *in situ*.

In the present instance the bronchiectasis was primary. The patient had had chronic bronchitis for many years before any evidences of phthisis made their appearance. The same may be said of two other cases I have had lately under observation. In both of these the bronchiectasis was bilateral, as every pulmonary lesion should be that is consequent upon bronchitis. In one of them, a middle-aged colored woman, the signs and the *post-mortem* revealed coexistent fibroid change. In the other one, living yet, I do not feel sure of the existence of fibroid phthisis. In these three cases the dilatation was more marked in the upper lobe of the lung; at least the tendency to excavation of the dilated tubes has been greater at the upper lobes in the five cases of bronchiectasis with cirrhosis that I have noted. Of two of these I remember but little beyond the point just mentioned, and that the pulmonary symptoms developed in the course of Bright's disease (large fatty kidney). The renal symptoms became almost latent after the evidences of phthisis became well marked.

About the relations of cause and effect between cirrhosis of the lung and bronchiectasis, it may be said that they are mutual. Few cases of chronic bronchitis terminate in pronounced dilatations, and in these few cirrhosis is very apt to develop, as a direct consequence of the collapse of surrounding lung-tissue, and also of the inflammatory action going on in the walls of the tubes. It is well known that pulmonary caverns are generally surrounded by fibrous-tissue walls. It seems to me that the dilated bronchi act the parts of old cavities disseminated throughout the lung, thus giving rise to a diffuse and bilateral cirrhosis. So in the present case this is not rarely accompanied with epithelial accumulations in the air-vesicles. On the other hand, cirrhosis as a primary disease is rare, whether due to pneumonia, pleurisy, old age, syphilis, heart-disease, or the inhalation of certain irritants. Whatever its cause, it frequently terminates in bronchial dilatation. In this manner one condition is as frequently primary as the other. It is in the latter class of cases, especially when due to chronic pleurisy, that cirrhosis and bronchiectasis are more apt to be unilateral. It is of interest to note in this case the probable connection of the bronchitis with typhoid fever. I also wish to state that we can exclude syphilis, heart-disease, anthracosis, and siderosis, from consideration in the present case. *February* 14*th*, 1878.

7. *Metastatic abscess of lungs following severe wounds of scalp, cranium, and buttocks.*

Presented by Dr. E. O. Shakespeare.

The specimen was obtained by a *post-mortem* examination made for the coroner on the body of Patrick Murray, at the Presbyterian Hospital. I know nothing of the history of the case, except from inquiries of the resident physicians of the hospital, who were present at the *autopsy*, one of whom has kindly furnished me with the date of the subject's admission, and a temperature-sheet, which presents some peculiar curves:

"Presbyterian Hospital, February 12th, 1878.

"Dear Doctor,—The following is a concise history of the case we posted some time ago. P. M., aged 20, white, laborer, single; personal habits good; was admitted on the evening of December 20th, 1877. He fell forty-five feet, striking his buttock against a bucket which was coming up. The bucket turned him, and he fell ten or fifteen feet farther, striking upon his head. Examination revealed a lacerated wound of the scalp about one inch in length over the occipital region, extending down to the bone. The periosteum was torn off for a space of a half-inch. Found no other lesions of the head. The buttocks were contused. The left had a deep wound extending into the muscular tissue. The right buttock was greatly ecchymosed. The temperature-sheet which I enclose is very remarkable, even for a pyæmic case, and will be of more interest to you than anything else I could send:

"Dec. 29th. M. 102½°; E. 103½°.—Dec. 30th. M. 102°; E. 104½°.—Dec. 31st. M. 104½°; E. 106½°.—Jan. 1st. M. 105½°; E. 104°.—Jan. 2d. M. 104½°; E. 105½°.—Jan. 3d. M. 104°; E. 106½°.—Jan. 4th. M. 103°; E. 104½°.—Jan. 5th. M. 105°; E. 106°.—Jan. 6th. M. 105°; E. 107½°.—Jan. 7th. M. 104°; E. 103½°.—Jan. 8th. M. 103°; E. 105°.

Very respectfully,
W. C. Hollpeter, *Res. Surg.*"

It may be stated, in addition to the above, that, notwithstanding the distance fallen, and the injuries to the head, there were no marked head-symptoms; that nearly ten days after injury the patient began to exhibit symptoms which soon developed into those of a marked pyæmia, from

which he succumbed nineteen days after injury. The following is a brief abstract of the notes of the *autopsy*, made January 9th, 1878:

External examination.—A purplish line under each eye; slight ecchymosis of bulbar conjunctiva; pupils slightly and equally dilated; healing lacerated wound of left buttock, about an inch deep; cannot run probe below its bottom. On back of head, little to right of median line, an incised wound of scalp, partly healed, one inch and three-quarters long, edges slightly swollen; at bottom, bone denuded of pericranium; could not feel fracture or depression by using end of finger.

Internal examination—Head.—Numerous extravasations of blood into deep layers of scalp, and between latter and skull, extending almost over whole cranial vault; this more marked around seat of external wound. Upon first glance, seemed to be no break in surface of bone at seat of wound, but close inspection showed two faint, narrow, short cracks, and a scarcely perceptible depression. Opposite this, inner table showed stellate fracture with some depression of inner plate of right parietal bone. In outer plate of left parietal, a small, fine linear fracture; opposite, in inner plate, a stellate crack. In the frontal bone, commencing at coronal suture, one inch to right of median line, and extending to right superciliary ridge, a sharp, gaping linear fracture through the whole thickness of skull; gaping line of latter fracture filled with blood and lymph. This material was found in very thin layers on inner surface of calvarium, around the region of the fracture. Dura mater, everywhere readily separable from calvarium, was covered along median line by thin layer of blood and lymph; membrane not adherent to pia mater, nor was it inflamed; inner surface apparently normal. No marked changes in other membranes, nor in cerebrum or cerebellum.

Thorax.—Some inflammation of anterior mediastinum.

Right Lung.—Tolerably recent adhesions to wall of chest, particularly near apex, and posteriorly. No effusion into pleural sac.

Left Lung.—Bands of adhesion more numerous, were mainly lateral and posterior, binding lung firmly to thoracic wall. Some straw-colored effusion in left pleural cavity, in it flakes of lymph and pus; surface and substance of each lobe of both lungs studded with numerous circumscribed abscesses, varying in size from that of a pea to that of a walnut, some round, others wedge-shaped, others between these two forms; when at surface, latter raised; pleural covering here thickened; section through abscesses showed them to be surrounded by an area of granular hepatization, beyond which was another zone of intense congestion; lung-tissue between these abscesses crepitant, but much congested, par-

ticularly posterior portions; abscesses rather more numerous in left than in right lung.

Heart.—Chicken-fat clot in right ventricle, sending prolongation beyond branches of pulmonary artery; organ otherwise normal, as were also large vessels.

Abdomen.—*Liver* normal. *Spleen* showed a subcapsular rupture of pulp in a line extending one inch with surface. *Right kidney* slightly congested, otherwise normal; ureter and bladder normal. Around left kidney, which itself was not unhealthy, a large perinephritic collection of pus; infiltration of subperitoneal connective tissue extending from spleen to left groin; left psoas muscles softened and infiltrated with pus; probe could not demonstrate any connection between this collection of pus and the wound of buttock, although lymph-glands opposite its location were enlarged and purulent. No involvement of joints could be determined.

February 14*th*, 1878.

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8. *Embolic pneumonia concurrent with disease of the heart.*

Presented by Dr. E. O. Shakespeare.

I am indebted to Dr. O'Neill, resident physician of the Philadelphia Hospital, for the following brief summary:

Charles H., aged 42 years, a tobacco-merchant; had always been addicted to the use of tobacco,—an inveterate smoker, and somewhat of a chewer; had enjoyed perfect health up to about three years ago, when he had a stroke of paralysis, the right side being affected. He entered the Philadelphia Hospital on the 29th of January, 1878. On auscultation there was heard a decided murmur with the first sound of the heart, at the apex and left side. There was a bounding pulsation and jerking pulse at the wrist. There was œdema of the lower extremities, extending far up the legs. He suffered with dyspnœa, so that the sitting posture was constantly retained. Articulation was slightly impaired.

Diagnosis.—Hypertrophy, with dilatation, and a button-hole mitral valve. Death was sudden, occurring in from five to eight minutes.

Post-mortem examination.—*Appearance of the body.*—Face and neck cyanosed; legs and feet swollen. On opening the thoracic and abdominal cavities there was found a straw-colored serous effusion.

Heart.—Slightly dilated left auricle; hypertrophy of left ventricle, with constricted button-hole mitral valve. *Ante-mortem* clot in the left auricle. No clot in right heart.

Brain.—Softening towards the base of left hemisphere; an old cyst, the contents of which had become fluid, was found below floor of left ventricle, about an inch behind the anterior horn, and the same distance to left of median line.

Lungs.—Right lower lobe shows three or four infractions, varying in size from that of a hazel-nut to that of a walnut. They extend to surface of pleura, are mainly wedge-shaped, and present, on section, a red, granular appearance, are solid, and have sharply-circumscribed borders. Around the border is a zone of intense hyperæmia. Only one shows any tendency to softening. There is a small similar infarction in the middle lobe; also one in the upper. At lower anterior edge of left upper lobe are two infarctions; none elsewhere in left lung. Pleural surface of all the infarctions is raised, and there are some bands of lymph here. In one of the infarcts a firm, solid, reddish clot was found occluding the lumen of the largest vessel in it. The lung-parenchyma elsewhere was somewhat hyperæmic, but otherwise normal. Liver, spleen, and kidneys, not abnormal. *February* 14*th*, 1878.

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9. *Miliary tuberculosis with pleuro-pneumonia.*

Presented by Dr. E. O. Shakespeare.

Dr. Garrett, resident physician of the Philadelphia Hospital, furnished the following abstracts of the clinical history of the two succeeding cases:

Walter M., æt. four months and six days. Parents are healthy, and, as far as can be ascertained, have no syphilitic taint. The mother is 23, and this is her fourth child, each being by a different man. All her children are dead. The first died with symptoms of jaundice. The other two were taken with bronchitis, and died with the same symptoms, the mother says, as the case before us. Baby Walter was a large, plump, and healthy child until this last attack of sickness. He was taken sick in the middle of December with a cold in the head, which passed into bronchitis, and then a few days later into a pleuro-pneumonia. During the last two or three weeks of his sickness he lost flesh, but not to a very marked degree. His symptoms were cough, which varied at times in severity; fever, which was severe at night, and almost entirely disappeared in the morning; pain, at times very severe, shown by a very peculiar distressed cry, great restlessness, a rolling of the head from side to side so that the hair was worn from the back of the head, a contraction of the

eyebrows, a pulling and twisting of the fingers, etc.; and a tendency in the bowels to be loose. The last week was marked with occasional convulsions.

Treatment consisted in counter-irritation and warm moist applications to the chest; stimulating and sedative cough-mixtures; and supportive and anodyne measures.

Autopsy.—Brain.—Slight fulness of large vessels of pia mater, which nowhere showed decided milkiness or deposits of lymph. On vessels in fissura Sylvii, probably a half-dozen minute miliary tubercles. Otherwise appearance of encephalon and its membranes not abnormal.

Lungs.—Scattered through substance of all lobes of right lung are numerous minute, firm, gray nodules, presenting the appearance of miliary tubercles. Around these are small zones of lobular pneumonia. Intervening portions of lung more or less congested; slightly crepitant. Left upper lobe shows only a few of the above-named points. In left lower lobe they are more numerous than in last-named lobe, but not so abundant as in right lung. No decidedly cheesy points. The bronchial glands are enlarged, some of them red, others gray, but not softened. Bronchial and tracheal mucous membranes not much affected. Over whole left lung pleuritic membrane thickened and covered with thick layer of lymph.

Heart.—Normal. Liver and spleen show numerous disseminated small gray tubercle-like points. Mesenteric glands somewhat enlarged, but not softened. Some of them present gray surface on section. Kidneys normal. *February* 14*th*, 1878.

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10. *Chronic catarrhal pneumonia.*

Presented by Dr. E. O. Shakespeare.

Elizabeth J., æt. three months and twelve days. Was unable to obtain any antecedent family history. Mother died nine days after she was delivered, of traumatic peritonitis, consequent upon rupture of the uterus. She was known to have a cough, but it is not known whether she had phthisis. Child at birth was fat and healthy-looking, and weighed eight pounds. At the end of its first month it began to emaciate, and continued to do so until death. She had no cough until one month ago, when she had an attack of bronchitis. Since this attack she has had daily evening exacerbations of temperature. During the last week she had occasional attacks of epistaxis.

Autopsy.—Brain, heart, liver, spleen, intestines, kidneys, apparently normal.

Right Lung.—Upper lobe, apex, and posterior portion atelectatic; surface depressed, dark bluish-red; leathery consistence. Small portion of upper posterior part of lower lobe in same condition, as well as a few small spots at upper anterior part of middle lobe. Elsewhere over the surface of the lung were scattered here and there small gelatinous-looking spots, with their surface raised above the general level. With the exception of areas corresponding to the above-named points, surface of lung was smooth, light pinkish color; its substance was finely crepitant. Bronchial glands on this side slightly enlarged and red. Bronchial mucous membrane not visibly altered. No pleuritic adhesion on the side.

Left Lung.—Firm adhesions at apex. Both lobes solid, not much swollen. Upper more firm than lower. At apex of upper lobe is a large, irregular cavity as large as a walnut, partly filled with a gray grumous fluid. Thickened pleura forms outer wall of cavity. Here is the location of the adhesions. The surface of section of the upper lobe shows a firm, somewhat semi-transparent pinkish-gray infiltration of parenchyma, scattered over with numerous yellowish cheesy points a little larger than a mustard-seed. Some of these points have softened centres. Cut surface of lower lobe shows same general appearance. Cheesy points not so numerous. Lymph-glands at root of this lung large, cheesy, and softened. Pus in smaller bronchi.

Thin sections of lungs from first case (*miliary tuberculosis*) show, under the microscope, small tubercles, mainly on small bronchi. Perivascular sheath of vessels but seldom involved. Around tubercles are areas of catarrhal pneumonia. Portions of lung between nodules show slight irritation of alveolar epithelium, and fulness of corresponding capillary nets.

Thin sections from the left lung of the second case (*chronic catarrhal pneumonia*), under the microscope, show the whole of the lung-substance in a condition of catarrhal pneumonia of the second stage. Some few of the cheesy nodules do not differ in appearance from those of the first case. Right lung was not examined microscopically.

February 14*th*, 1878.

Dr. Guitéras said he should have presented at the last meeting a specimen of thrombosis of the pulmonary artery, with small areas of catarrrhal pneumonia. The subject is sufficiently related to that of the present dis-

cussion to be considered to-night. A middle-aged woman was admitted to the wards of the Philadelphia Hospital suffering with capillary bronchitis. Careful percussion demonstrated the existence of small patches of consolidation at the right apex. She died of apnœa three or four days later. The symptoms of bronchial obstruction had diminished, but the pulse was very feeble and frequent. She did not die of œdema of the lungs. The *autopsy* revealed the existence of the lesions diagnosed. The lobules consolidated were four or five, and scattered. The catarrhal consolidation was very recent. The right heart was distended with blood. The clot was not large, but when traced into the branches of the pulmonary artery it lost its chicken-fat appearance, and became of marbled-gray and black color, and rotten and friable. He discovered no disease of the walls of the pulmonary artery.

Dr. Alibut has recently advanced a very ingenious theory to explain the symptoms of uræmic asthma. He suggests a state of spasm of the pulmonary arterial system; so that the dyspnœa, he thinks, is due to the failure of the blood to reach the air, and not to any obstruction to the movements of the latter. Dr. G. suggested the adaptation of this theory to his case, and he called attention to this point, because it is very possible that this condition of thrombosis and consequent dyspnœa resulting from a deficient supply of blood to the air-vesicles is frequently overlooked.

On the same day of this *post-mortem* he was called in consultation to a case of membranous croup. After many hours of dyspnœa and cyanosis, the child died, without one single râle of œdema of the lung making its appearance. He should say also that there were no evidences of blood-poisoning; he meant no diphtheria.

In regard to the specimens before the Society, he desired to call attention to their significance, as they illustrate beautifully the two forms of emboli that Cohnheim has studied so well. In the one case we have the mechanical embolus, with hemorrhagic infarctus of the area supplied by the vessel; in the other we have the infectious embolus, with abscess of the surrounding tissue. There are few observations in pathology less subject to exceptions than this: that the mechanical obstruction of a pulmonary artery never causes suppuration, and seldom gangrene of the parts beyond, and that an infectious plug will set up a suppurative process; in the latter case, from the nature of the process, a more or less spherical accumulation of pus is the result; in the other, a wedge-shaped infarctus.

It is a curious fact that a stoppage of the flow of blood in an artery should cause hyperæmia and hemorrhage of the parts beyond. Ludwig suggests that the obstruction is not complete, and that the slowing of the circulation

in the capillaries causes the blood-corpuscles to aggregate, so that a terminal obstruction is produced; the further ingress of blood, in spite of the embolus, increases the tension, and a ruture supervenes. Cohnheim thinks that the congestion is due to a back flow of blood from the pulmonary veins, the pressure in the arterial side having been reduced to a minimum by the embolus. Cohnheim objects to introducing the anastomoses with the bronchial arteries as an element in this hemorrhagic process. This is a theory that had occurred to him (Dr. G.), and he was not willing to give it up. The anastomoses between these two sets of vessels are terminal in position and capillary in character. It is upon the latter fact, viz., their size, that Cohnheim establishes his objection. He (Dr. G.) failed to sée sufficient tension in the backward flow to rupture the vessels and cause the hemorrhage. The blood in the bronchial arteries is under greater tension, which probably widens the channels to establish a collateral circulation in the affected part. It must be remembered also that this blood forced into these vessels is not the blood that they are adapted for; it is arterial blood; hence another source of hemorrhage, that will remind us of the numerous hemorrhagic spots found in the lung after transfusion of arterial blood.

Dr. G. said that no one who had had opportunity of observing cases of mechanical emboli could fail to observe the absence of suppuration in these cases, no matter what the condition of the patient was; often, in fact, very much broken down in the last stages of cardiac cachexia. The interesting fact mentioned by Dr. Norris, of the early conical shape of infectious infarctions, only proved that the plug acted mechanically before exerting any specific influence. In such cases Cohnheim even maintains that there must have been two emboli. He could not say that the dyspnœa in his case was all due to the thrombosis; but the symptoms of bronchitis (physical signs) had improved, and the *autopsy* did not show much obstruction in the tubes. The patches of catarrhal pneumonia were so small that they were undetected at first.

He agreed with Dr. Shakespeare that very few cases of obstruction of the pulmonary artery had been studied in the light of the modern ideas about tuberculosis. In fact, it is difficult to determine from the accounts of authors whether the phthisis observed in such cases is tubercular or not. He believed that there was no doubt as to the fact, that ischæmia favored the development of those slow processes that led to destruction of pulmonary tissue. Hence their frequency at the apices of the lung, and the truth of the theory advanced by Peter, viz., that phthisis is rare in disease of the left heart, because, the dependent portions of the lung being

in a state of constant engorgement and comparatively inactive, the upper portion are, of course, the seat of a compensatory activity.

Dr. Nancrede remarked, with regard to the shape of the infarction, that he thought we looked too much in a pathological way—or, if he might term it so, in too mechanical a way—at this subject. In the present cases he thought that we could see triangular infarcts in the pyæmic case, and rounded ones in the embolic one, thus showing that the rule was not at all an absolute one. The consideration of the systemic condition was too much overlooked. The present specimens, it seemed to him, bore out this view. In the pyæmic one we have a man suffering from several severe injuries,—one of them of the skull and brain,—with suppuration lasting several weeks, his system broken down and prone to suppurative trouble. Now, inflict any kind of injury upon his lung, and suppuration would be likely to ensue. If any surgeon were unwise enough to amputate a limb during the course of pyæmia, would he have any healing occurring? Quite the reverse; he would only encounter unhealthy suppuration. Again, in the embolic specimen we have a case in perfect health, comparatively speaking, whose lungs are suddenly extensively plugged. What is the result here? Manifestly such an interference with the functions of organs essential to life that death results before suppuration has had time to occur. Dr. N. believed that if time were allowed, mechanical emboli would cause suppuration in certain cases. The reason it is not seen is that where extensive enough to allow us a *post-mortem* examination, death is rapidly produced by overwhelming injury to a vital organ. He had risen with diffidence to express opinions in any degree opposed to the authorities quoted, but he must protest against the one-sided, mechanical, and as he thought, in consequence, unscientific way in which the problems of disease were nowadays treated.

Dr. W. F. Norris said, in reference to the form of the abscesses, that it had been his lot to see many such cases while in the army, and he thought the shape of the patches was due to the stage of the disease when the *post-mortem* was made. The majority are found near the surface of the lung, and in the early stages are wedge-shaped, with the base presenting outwards. It is usual to find in the same organ several such patches in different stages of development, and there is a tendency to become round when suppuration takes place. He had, however, seen infarctions which had already the greenish-yellow hue of pus, and were almost ready to lose their solid consistence, still retaining their pyramidal form.

Dr. Henry thought that, in attempting to account for the presence or absence of suppuration in cases of embolic pneumonia, constitutional con-

ditions were not to be excluded. The influence of cardiac disease upon inflammatory processes in the lungs is universally admitted, but it is perhaps not so well known that this influence may extend to other processes. According to Lebert, there is in congenital stenosis of the pulmonary artery a remarkable predisposition to the deposit of miliary tubercle in the lung; while in venous stasis dependent upon mitral disease, miliary tubercle is so rare that this form of cardiac disease is regarded by some as inimical to its deposit. If, therefore, cardiac disease can so powerfully and variously influence morbid processes in the lung, it is highly probable that other conditions of the system affecting the circulation may have similar though less powerful effects.

Dr. Henry remarked, that if Dr. Guitéras intended to signify that there was an *arterial* anastomosis between the bronchial and pulmonary arteries, on the distal side of the points of infarction, he could not agree with him in the idea that such an anastomosis would favor the morbid process. In his opinion it would have precisely the contrary effect, by favoring the return venous circulation. Cohnheim's theory of infarction applies exclusively to *terminal* arteries, that is, to an artery, or rather to a portion of an artery, beyond which there is nothing but capillary vessels.

Dr. Shakespeare thought with Dr. Guitéras, that the explanation of sudden dyspnœa occurring in other states besides that existing in so-called uræmic asthma, might not infrequently be found in an obstruction, embolic or otherwise, to the free course of the blood in the branches of the pulmonary arteries, rather than in an impediment to the air in its passages,—a failure of the required quantity of blood to reach the air at the proper place, rather than the curtailment of a necessary supply of air.

He could not, however, agree with Dr. Guitéras in the support which the latter had given to the opinion advanced and defended by Rindfleisch, that the bronchial artery, through its sparse capillary anastomosis with the small branches of the pulmonary artery, furnishes the blood which engorges the area of infarction. He was constrained to think, that the objections which Cohnheim had raised against this hypothesis were conclusive and insurmountable. Not only is the anastomosis relied upon by Rindfleisch extremely limited, but it is mainly of a capillary nature; and so far as the small terminal branches of the pulmonary artery are concerned, it can scarcely be said to exist at all. It is just in these small terminal branches where the embolus which gives rise to the infarction usually lodges. So that in many cases, if not indeed in the majority, it is not probable that even this very sparse capillary anastomosis can be said to exist. An arterial communication is generally out of the question; but

were the anastomosis to be of that character, *a priori*, it would seem that there could then be no infarction, or at least only a very minute one, if any at all. As to this point he therefore coincided with Dr. Henry. He endorsed in general the conclusions as to the course and modus of an infarction, reached by Cohnheim after a thorough experimental study of the subject; and in particular the regurgitation of blood from the venules connected with the capillary radicles of the obstructed artery as a competent cause of the engorgement and extravasation of blood in the infarcted area.

Dr. S. also admitted, that in our study of the embolic processes we should not overlook general conditions, any more perhaps than we should neglect them while examining any other morbid process. One cannot deny a very important influence to the action of the heart, and the state of the blood in modifying or even originating morbid processes in different parts of the lungs. The observation by Peter, of the extreme rarity of phthisis pulmonum where disease of the left heart, resulting in mitral regurgitation, had previously existed, quoted by Dr. Guitéras, and the declaration of Lebert, that in congenital stenosis of the pulmonary artery there is a remarkable predisposition to the formation of tubercle in the lung, instanced by Dr. Henry, are extremely forcible reminders of that important influence. Nay, more, they may even be regarded as the finger-posts which point towards the cause and the cure of this fatal "ill which flesh is heir to." Men may question the practical utility and value of pathological research. Although the nature of tuberculosis is only to be read to-day through the veil of many lingering clouds which must be removed, and the study of that nature has been, and still is one of the most difficult and complicated in the whole field of pathology, yet, thanks to the investigations of the pathologist alone, besides many other points in its natural history which have been settled, it has been demonstrated that the disease can only exist when the blood-supply is far below that which is needed, and that the first step in the direction of a cure, or the healing of a tissue where tubercle is deposited, is the production around the morbid mass of a marked inflammatory hyperæmia. One of the essential characteristics of tuberculosis is that it is a bloodless process. Any condition, therefore, which tends to produce, or to continue, or to aggravate an ischæmia in a scrofulous or tuberculous person, tends to the production or the aggravation of the disease. On the other hand, the opposite condition is a powerful antagonist to the production or the continuance of the malady.

Dr. S. was willing to go as far as reason and experience would lead any

one, in according to general conditions a due amount of consideration, but he thought that such guides would not, in the present state of our knowledge, permit him to take such advanced ground upon the pyæmia question as Dr. Nancrede had assumed. He had no intention, however, of entering upon any lengthy discussion of the nature of pyæmia. His object in presenting the specimens had been merely to exhibit for the benefit of those members of the Society to whom the opportunity seldom or never comes of examining and studying for themselves the differences generally to be seen by the naked eye, between embolic or non-infectious pneumonia, and the pyæmic or metastatic abscess, arising from an infectious embolus lodged in one of the vessels of the lungs.

He had never seen specimens which better contrasted the naked-eye appearances of those two different sequences of embolism in the lung, and he could not at all agree with Dr. Nancrede, when the latter stated there was no essential difference between the form or shape of a pyæmic abscess and that of the infarction from a non-infectious embolus, and referred to the exhibited specimens as a corroboration of his opinion. On the contrary, the specimens show, in as typical a manner as possible, the difference which generally prevails.

Dr. S. did not believe it to be by any means an absolute rule that in embolic pneumonia the infarctions are wedge-shaped, with their base on or towards the pleura, and that in the metastatic abscess of pyæmia the infarctions have rounded borders, but he thought that generally this distinction was to be observed. In the present cases it can be very distinctly seen that nearly all the infarcts in the pneumonic case have a general wedge-shaped outline, while almost all of the metastatic abscesses of the other case have rounded forms. Dr. S. believed, with Drs. Norris and Guitéras, that the original form of the pyæmic infarct is shaped by the same mechanical conditions, which generally secure to the non-infected infarction a pyramidal outline, which it retains, and that the inflammation and suppuration, which the irritating embolus of pyæmia rapidly induces, soon round off the corners and plane surfaces of the original wedge. When the *post-mortem* examination is made, the inflammation has generally existed long enough to have wrought this change. Hence the generally round or spherical aspect of the metastatic abscess. He believed also, that there is in pyæmia an essential tendency to suppuration, which cannot be satisfactorily accounted for by any amount of ordinary asthenia. Almost never, no matter how asthenic the patient may have been, do we find the infarctions of embolic pneumonia losing their characteristic shape from a secondary inflammation and suppuration. Dr. Nancrede attempts to escape

the force of this fact by saying, that we do not find the inflammation and suppuration here, simply because the patients are overwhelmed by the plugging of their lungs,—are drowned before the inflammation has had time to develop. It is a sufficient answer to this conjecture to state, that experience has shown that in the vast majority of cases which have been examined, the embolic patches are comparatively not very numerous, and are most frequently limited to the lower lobe of one lung, and that instead of death being caused by an overwhelming of the lungs, nearly all the patients die of some fatal intercurrent trouble. *February 28th*, 1878.

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11. *Syphilitic disease of the pleura and lung.*

Presented by Dr. W. H. Warder.

Thomas M., æt. 38, has always led an active life. Served in the army during the last war, and afterwards, one year in the army on the frontier. He came under my observation in January, 1873. At this time his general health was good. His weight was about two hundred pounds. He stated that in November, 1866, while on a visit to Rock Island, Illinois, he had an attack of pleuro-pneumonia, which was followed by empyema of the right side, which opened through the lungs, and large quantities of pus and blood were coughed up. So free was the expectoration at times as almost to strangle him. Two pints and a half by actual measurement would be thrown off in twenty-four hours. This expectoration continued through the winter and spring, getting somewhat better during the summer.

In the fall of 1867 his physician ordered him to Beaufort, South Carolina, to spend the winter. There he contracted intermittent fever in January, 1868, from which he did not entirely recover until July. His cough still continued, and did not disappear until the spring of 1869. His health from this time was good. He was married in the fall of 1869. About one year after, his wife miscarried at three months. The following year she miscarried at seven months. In January, 1873, I was called to attend her in confinement, and she was delivered of a still-born child, which had evidently been dead some days.

From the history of his wife's pregnancies, and she to all appearances being a healthy woman, I was led to suspect that the father had had syphilis. This, he acknowledged, occurred while he was serving in the army. Upon examination I found no involvement of the mucous membranes or

bones. There were some eczematous eruptions upon the lower extremities, and also upon the arms. I placed him upon specific treatment, which was done more in reference to his wife's miscarrying than to any manifestations for want of treatment upon his part. While under this treatment his wife conceived, and at full time was delivered of a living child weighing nine pounds. The child was healthy, and did well until two months old, when nodes and swellings showed themselves on the fingers, and bones of the forearm. These disappeared under constitutional treatment. The child at the present time is well grown and healthy.

Mr. M.'s health was uninterruptedly good until February, 1877, when he had a very severe attack of bronchitis with congestion of the right lung. He entirely recovered from this attack, but very soon afterwards pustular eruptions made their appearance upon his feet and legs, some of these pustules assuming the form of abscesses, discharging quite a quantity of pus, and about the ankles degenerating into characteristic ulcers. There was general derangement of the secretions, with slight fever in the afternoon and evening. Under the use of pil. hydrarg., quinine, and potassium iodide, his general health was restored, but the pustules did not entirely disappear, although they were very much better.

In May he moved to Bound Brook, New Jersey. He was not under my care until November 16th. He stated that in the early part of August he had taken a very severe cold, with chill, followed by high fever, and was treated for remittent fever by his attending physician. From this time until I saw him, November 16th, there had been a series of improvements and relapses. There had been cough with slight expectoration, fever in the afternoon, and night-sweats. When I saw him in November he was suffering from a severe paroxysmal cough, with a tough, glairy mucous expectoration, with hectic fever in the afternoon and evening, and profuse night-sweats, difficult breathing upon making any effort in walking or ascending stairs, etc.

Pulse in the morning, 110; afternoon, 120.

Temperature.—Morning, 99½° F.; afternoon, 101½° F.

November 18th, Dr. Wm. Pepper met me in consultation. A careful physical examination showed that the respiratory movement over the lower part of the right lung was restricted, while that of the left lung was exaggerated. The heart's impulse was displaced towards the left, the apex-beat being in the line of the nipple. Resonance was good everywhere except over an area, semicircular in shape, which extended on the lateral aspect of the chest, from the liver-dulness below up to the line of the nipple, sloping downwards both in front and behind. Over this area there was

absolute dulness on percussion. Auscultation over this space gave only very feeble, scarcely audible bronchial breathing, without râles. There was also almost complete absence of vocal fremitus, and of vocal resonance. There were a few crackling râles in the lung-tissue immediately adjoining this area of flatness. Elsewhere the respiratory murmur was normal.

The above physical signs, viewed in connection with the general symptoms,—hectic fever and night-sweats,—with the history of the case, led us to the opinion that there was empyema, and we decided to explore the part with an aspirator needle. On the following day, the needle was introduced to the depth of two inches, in the seventh intercostal space in the line of the anterior border of the axilla. It passed into a solid, unyielding tissue, and no fluid followed.

From the syphilitic history of the case, and finding there was no pus, and the fact that there had been empyema in this side, we were disposed to think it probable that the pleura had become the seat of irritation with plastic exudation, which had become organized, and was pressing upon the lung, together with some syphilitic degeneration of that tissue. In the hope of securing the absorption of the deposit, we determined to place our patient upon the use of potassium iodide and hydrarg. bi-chlor., quinine, and opium, and with good nourishment, to wait and see if such a result would follow. Feeling certain that the disease was of syphilitic origin, the iodide was given freely, the quinine and opium three times a day, and one-sixtieth of a grain of atropia was given at bedtime to control night-sweats. Under this treatment the appetite improved, the hectic fever lessened, and the night-sweats almost disappeared. Daily exercise in the open air, when the weather was good. This course was steadily pursued until December 27th, with some slight improvement in general health; and now, finding that no absorption was taking place, but that the area of disease was extending, with no change in the physical signs, we determined to repeat the operation to see if any breaking down of the mass had taken place. This was entirely unsuccessful. Upon passing the needle into the mass it was found that the same condition remained as before. The area of dulness had increased, the breathing was more difficult upon exertion, the cough was more troublesome, and the displacement of the heart was much greater.

The iodide was increased, and the mineral acids with tonics were given to keep up the digestive organs. The chest was painted with tincture of iodine once a day. The cough was controlled by hypodermic injections of Batley's solution of opium at night. The patient was still able to

walk about, and each day, when the weather permitted, would take some exercise in the open air.

During January there was but little change in the case. The area of flatness gradually increased in every direction. It extended up into the axilla; it involved the posterior border of the lung, and also advanced anteriorly.

The apex and the anterior border were the parts which remained free last, and over them a remarkable tympanitic resonance existed for some weeks, until it was finally replaced by flatness, and the entire pulmonary area became non-resonant. It was to be noted, that as successive portions of lung were invaded, the only physical signs developed were large crackling râles, and then complete dulness with absence of respiratory murmur followed. The displacement of the heart steadily increased, until the apex-beat was at the anterior border of left axilla. There was at no time any valvular murmur. There were, however, a few dry clicking pericardial friction-sounds. The action of the heart was rapid but regular. As the case advanced (about February 1st) there appeared enlargement of the lymphatic glands of the right axilla, and soon afterwards of the supra-clavicular glands. The enlargement ultimately became quite great. About March 1st difficulty of deglutition appeared and slowly increased, so that for two weeks before death he was only able to swallow small quantities of liquid. The breathing became more and more difficult, and somewhat stridulous, and the voice feeble. The cough became looser, and for several weeks there was considerable purulent expectoration. He had several attacks of diarrhœa, which made it necessary to stop the use of the iodide of potassium and mercury.

A third paracentesis was performed about February 25th, at the earnest request of the patient and of his friends. There was no expectation of finding liquid, and in fact the needle, though introduced to the depth of two and one-half inches, failed to reach any. It was then determined to use frequent injections of liq. iodine comp. into the pulmonary tissue by means of a hypodermic syringe, introduced through the intercostal tissue. These injections were continued daily, or on alternate days, for six weeks. They caused little pain or irritation. Death finally occurred after a protracted and painful struggle, with symptoms of cerebral disturbance, on April 8th, 1878.

Post-mortem examination.—Head.—Not examined.

Thorax.—Left lung normal size. Presented some friable adhesions. Tissue deeply congested and crepitant throughout, and presented, in the posterior part of the upper lobe, nodules similar to the masses found in

right lung. The glands about the bronchus were diseased and much enlarged. Right lung much enlarged and extending forward into anterior mediastinum, and very strongly bound down by adhesions, so that it was very difficult to remove. On removal the mass presented the general contour of the lung, so that it was evident in large part it consisted of lung-tissue, infiltrated with morbid growth outside of the lung-tissue. Again, beginning at the base of the lung, there was a transverse line of demarcation, which indicated the apparent division between the lung substance and large mass of extra-pulmonary origin.

On making sections deep into the lung substance, no trace of pulmonary structure was found. The mass was dense, and presented a yellowish-gray color, and of a firm fleshy consistence intersected by fibrinous trabeculæ. At points corresponding to the punctures, minute points of softening stained by iodine were found. In the centre of the lower lobe a cavity of considerable size filled with shreds of tissue and pus, lined by a shaggy wall, existed. The size of the cavity was at least two and one-half inches in diameter. The division between the lower and middle lobes was only in part demonstrated. Through the lower and middle lobes the morbid tissue formed a uniform mass. In the upper lobe the lung substance was replaced by an aggregation of firm nodules of varying size. In a few places there were nodular formations on the exterior of the pleura, or at least a uniform fibrinous membrane could be dissected up; and in a few places, especially on the upper lobe, it brought with it nodules springing from the pulmonary surface.

The anterior mediastinum was occupied by a dense mass, tightly adherent to the anterior border of the right lung. The mass, which was partly composed of diseased glands, and partly of infiltrated connective tissue, was incorporated with the right side of the pericardial sac. On opening the latter membrane no adhesions were found to exist, only in a few points on the reflected layer, where there were small fat nodules elevated above its surface. On the other hand, there were some growths on the external surface of the heart, two of which were of considerable size, the largest being almost two inches in diameter, and elevated at least one-half inch above the surrounding level of the heart. On section it presented structure similar to the pulmonary growth, and evidently involving the muscular tissue of the heart. The glands of the posterior mediastinum were much enlarged.

The liver was somewhat enlarged, congested, and soft. There appeared to be no albuminoid degeneration.

The spleen was apparently normal.

The left kidney contained a secondary deposit, resembling a gumma, about five-eighths of an inch in diameter.

The stomach and intestines appeared healthy. No peritonitis, and no peritoneal growths. Some of the mesenteric glands were enlarged.

Nodules of varying size were found in the subcutaneous tissue of thoracic and abdominal walls. These, together with the enlarged cervical and axillary glands, were hard, and on section presented a pale yellowish-gray color, and a dense fibrous structure.

The case, so far as I know, is unique and full of interest.

May 23*d*, 1878.

Report of the Committee on Morbid Growths:

"A microscopic examination of a thin section, taken from the mass which occupies the position of the right lung, shows it to consist almost entirely of a dense fibrillar connective tissue. The blood-vessels, which are few in number, are surrounded by an infiltration of indifferent cells. One of the small nodules taken from the left lung, upon examination, is found to consist of a similar tissue in an earlier stage of development. The alveolar spaces in the proximity of the nodule are seen to have their walls thickened by fibrillar connective tissue. The cells lining the alveoli are in a state of proliferation." *June* 27*th*, 1878.

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12. *Cancer of the pleura and uterus.*

Presented by Dr. Wm. Darrach.

A. W., æt. 40, married, white, was admitted, March 27th, 1877, in the Germantown Hospital. Breast removed by Dr. Müller; wound healed by granulation.

Re-admitted December 3d, 1877. Cicatrix and enlarged axillary glands removed also by Dr. Müller.

Re-admitted third time, April 11th, 1878. Died May 14th, 1878.

At the time of her admission she complained of a great deal of pain and weakness. She was confined to bed. There was considerable dulness on percussion posteriorly, on the right side particularly, and some on the left.

Temperature ran from 99° F. to 100° F.

The principal symptoms were pain and dyspnœa.

Post-mortem, thirteen hours after death.—Examination showed the following appearances and condition:

The body was somewhat emaciated. Over the left mammary region there was a large cicatrix, instead of a gland, which had been removed some fourteen months before death. This cicatrix and neighboring region were interspersed with cancerous nodules.

Upon laying open the integument covering the chest, a layer of subcutaneous fat, yellowish in color, one-half inch thick, presented itself. The muscular fibres were normal in appearance.

Under the tissues of the left mammary region cancerous tissue adherent to the ribs presented itself, involving the periosteum.

The sternum and portion of ribs being removed, three pints of fluid, greenish yellow in color, were found in the *right* pleural cavity.

The *right* pleura presented some adhesive bands, and a mottled appearance, with some *nodules.*

The *left* pleura at the base was studded with small *cancerous* nodules, and contained one pint of fluid.

The costal pleura presented the same mottled appearance. The lower portion of lung adherent to the diaphragm.

Lungs.—Right much contracted and drawn to the upper portion of the cavity; crepitant and congested. When cut across, a *creamy fluid* was seen in some of the bronchial tubes, but *no nodules* of *cancer* could be detected in the lung structure.

The *left lung* was adherent to the diaphragm, and congested but crepitant. Here also there was an absence of cancerous nodules.

Heart normal in size and appearance; whitish fibrinous clot in left ventricle.

Stomach normal.

Liver full size. Several whitish nodules presented themselves, about the size of a pea, on the surface and somewhat imbedded in the substance, but none could be detected when the liver was *cut into*, as I have seen in other cases.

The *kidney* presented no abnormal appearance, except one nodule.

Uterus normal in size. A round deposit appeared outside, and upon being laid open, a whitish round nodule, about one-fourth of an inch in diameter, showed itself in the right cornu.

The *spleen* was congested, and contained a nodular mass on the surface, and extending into the substance. The nodules in these various organs presented the characteristic cancerous appearance.

No one organ was sufficiently affected to interfere with its function, except the lungs.

The immediate cause of death seemed to be the effusion in the pleural cavity.

The case was of interest to show how general the disease manifested itself in almost every organ except the lung, stomach, and glandular structure of the bowels, and also the choice of integument, and the membrane investing organs not in the deep tissue. *May 29th*, 1878.

Report of the Committee on Morbid Growths:

"A microscopic examination of the nodule situated in the walls of the uterus shows it to be composed of fibrillar connective tissue, arranged in such a manner as to form alveolar spaces, which spaces contain large nucleated epithelial cells, demonstrating the new formation to be carcinomatous in structure, variety 'schirrhus.' Sections made from one of the nodules existing on the pleura proved by examination to consist of a similar new formation to that found in the uterus." *June 27th*, 1878.

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13. *Embolic pneumonia.*

Verbal communication by Dr. Guitéras.

Dr. Guitéras desired to relate very briefly a case bearing upon the discussion of embolism. He had found at the Philadelphia Hospital a pair of lungs presenting numerous small patches of gangrene. He had immediately suspected the existence of some focus of infection, since the tissue between the foci was healthy. On asking, it was discovered that the man had had during his rather short illness a dark and fetid discharge from one ear. The body was placed again upon the table, and the skull was opened. A portion of the petrous portion of the temporal bone was found broken down, and the lateral sinus near it in a condition of grangrene; its walls and the contained clot presented a greenish hue, and were fetid. The body had not commenced to decompose. The emboli washed from this focus was evidently infectious, and they gave rise to a process similar to the one taking place at the fountain-head. The patient was a robust adult. *June 27th*, 1878.

V.—THE GENITO-URINARY ORGANS.

a. KIDNEYS, BLADDER, ETC.

1. *Cystic suppurating kidneys.*

By Dr. C. B. Nancrede; notes by Dr. Fairfax Irwin.

This man, æt. 75, admitted August 6th, 1877, has experienced a difficulty in passing his water for the last nine months. He says it comes away in very small quantities at a time, and with much straining. Says he has not had a stool for nine days. There seems great pain on pressure over the abdominal region, which is swollen and tumid. Face is flushed; pulse full and rigid, 65 per minute; the arteries have an atheromatous feel; heart-sounds are weak; first sound more valvular than natural; second sound accentuated. Cannot pass a metallic catheter, but succeeded in passing a No. 5 flexible catheter; drew off half a pint of urine. There is evidently a great enlargement of the prostate gland.

August 7th.—Better to-day; bowels slightly opened; catheter used.

August 8th.—Has had no passage; drew off about one quart of urine with catheter.

August 9th.—Has had three stools.

August 10th.—Worse to-day; suppression of urine; passed a stomach-tube up the rectum to sigmoid flexture; little result.

August 11th.—Has not passed more than a drachm of urine; passages very liquid; steadily grew worse, and died at half-past three A.M.

August 12th.—Post-mortem examination.—Rigor mortis well marked. Hyperstasis of posterior parts of the body. Heart enlarged; tricuspid stenosis; left ventricle, aorta, pulmonary artery, and vena cava ascendens contained large, firm *ante-mortem* clots. Both kidneys contained abscesses; the left a cyst also. Peritoneum inflamed. A few bands of old lymph were found constricting the ileum and rectum, thus accounting for the partial intestinal obstruction.

Dr. Harrison Allen said the mention of suppression of urine, and the difficulty of telling it from retention, interested him, and reminded him of

a case which came under his observation,—that of a woman who had been drinking, and had passed no urine for three days. He introduced a catheter, and was surprised to obtain but a very small quantity of perfectly fresh appearing urine. Her abdomen was large, and he thought possibly he had not entered the bladder perfectly and went for his case of catheters. He found, however, that he was right. Dr. Allen had asked himself the question why this should be. It occurred to him that there might have been an overloaded cæcum, which, pressing on the ureter, retained the urine in it for a time. So in Dr. Nancrede's case, could not the ureter have been compressed by the product of subperitoneal inflammation?

Dr. Nancrede said it occurred too soon after free micturition to make this a probable cause. He referred to a case in which the urine contained blood and a small quantity of albumen, followed by a peculiar train of symptoms, eclampsia, cough, fetid breath (whence gangrene of the lungs was suspected), and afterwards great abdominal pain. After death one kidney was found converted into a thin-walled sac, containing a large amount of thin fluid, with complete obstruction of the ureter. He thought it not improbable that the man had had old kidney disease, and pyæmia had supervened upon the passage of instruments.

Dr. Allen said that all are familiar with the fact that in dropsy diuretics will not act until the bowels are relieved by purgatives, because of the previous pressure on the renal vein. He thought his supposition in this case not impossible.

Dr. H. Lenox Hodge said that it would be very difficult for any growth or enlargement to press so firmly upon the ureters as to prevent the urine from entering the bladder. The attachments of the ureters are so loose, and the mobility so great, that they readily recede from the pressure of any enlargement. Their coats are so thick and strong as to resist great pressure. It would be almost impossible for any enlargement to compress a ureter on all sides at once, or to press it against the bony walls of the abdomen or pelvis. Urine would reach the bladder unless the pressure occluded both ureters at the same time.

Dr. Allen said this, in his judgment, did not apply to the ureter low down in the pelvis, where it lies in the female, at the side of the true pelvis, previous to the canal inclining downward and inward towards the bladder. As the ureter passes through the uterine plexus of veins near the cervix uteri, it is liable to be compressed by morbid products situated therein, and can even be lacerated in over-distention of the os. So that, in its pelvic relations at least, a comparatively slight cause might

squeeze the ureter against the side *of*, or compress it at some other point *within*, the pelvis.

Dr. Nancrede said there were old bands of lymph constricting the intestine, but the distention of the abdomen was almost entirely due to gas.

Dr. M. O'Hara thought, as to the opinion expressed by Dr. Allen, that the pressure of the column of fluid on the ureter, backed by the force of the blood, was very great, greater than any pressure from the abdominal effusion in ascites. We can form some idea of the amount of this *vis a tergo* from the enlargement of the bore of the ureter on the passage downwards of a large calculus, and we would feel assured that no mere weight of a passive fluid, pent up in the abdomen, would be able to overcome such a force.

September 10*th*, 1877.

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2. *Small calculus.*

By Dr. Nancrede; notes by Dr. Robt. Meade Smith.

R. D., æt. 4, admitted to children's surgical ward, Episcopal Hospital, September 5th, 1877, with vesical calculus. Robust, healthy-looking boy.

Examination of urine.—Pale amber color; slight yellow flocculent deposit; reaction faintly acid; sp. gr. 1015; a trace of albumen. *Microscopically.*—Pus corpuscles, few blood-disks, squamous and cylindrical epithelium; no casts or crystals; granular débris. After having had bowels well opened, with castor oil and enema, Dr. Nancrede extracted, by lateral lithotomy, an oxalate of lime calculus weighing sixteen grains. No accident or hemorrhage occurred in the course of the operation. The patient rapidly reacted from the ether which had been administered. Placed on milk diet. Some little oozing which occurred in the afternoon yielded to elevation of pelvis and application of cold. Slept all night.

September 10*th*, 1877.

3. *Cancer of the right kidney—Thrombosis of ascending cava and left renal vein.*

Presented by Dr. John Guitéras.

Henry F., a German, æt. 68. Admitted to the Philadelphia Hospital the 27th of February, 1878. His legs were swollen, hard, shining. Eight or ten ulcers, about the size of dimes, could be counted on each limb. They were surrounded by a dark purplish skin, showing no tendency to heal. The swelling extended up beyond the popliteal spaces. It was more marked in the left leg. He was anæmic, short of breath, without any thoracic lesion apparent. The gums were spongy, the teeth loose, his diet bad, his residence the out wards of the hospital, where scurvy abounds. As a case of scurvy, the man was treated, and improved much. He gained in vigor, in flesh, and in color. The ulcers healed, leaving some indurations. The right leg returned to a normal size, and the left was almost as much reduced. Now it was found that the horizontal position was necessary to keep the legs in this condition. If he stayed up they were sure to swell. Outside of this, and an occasional accident that will be mentioned shortly, he appeared in very good health, had very good color, and was the most cheerful patient in the ward; had no pain whatever; no albumen or casts in the urine. The belly was large, but nothing that might not be normal. The liver was large, and the superficial abdominal veins were somewhat distended. The heart was fatty, though not to a marked degree. An anæmic murmur at the base disappeared under treatment. While in the wards he had two severe attacks of hæmaturia, relieved by ergot and gallic acid. They left no trace behind.

The third and last attack commenced on the 20th of May. He was in bed, getting over a slight swelling of the legs, brought on by sitting up for two days. I did not see him at this time, but I gather from the notes of Dr. Roberts, the resident physician, that on that day the urine became dark and clotted in the vessel. On the 21st, "Passed no urine in twenty-four hours; bladder rose high up in the hypogastrium; passed a No. 20 catheter, and removed, by aspiration, about two ounces of clots. This was followed by the discharge of about fourteen ounces of bloody urine. In spite of astringents and hypodermics of ergotin the hæmaturia continued. On the 23d the hemorrhage was so great, and the condition of the patient such that, as a last recourse, Dr. Bruen suggested the injection of a weak solution of Monsel. The hemorrhage was controlled, but the weak pulse

did not improve, and he soon commenced to vomit. He continued to pass very small amounts of dark-colored urine."

May 26th.—I saw him, and found distinct symptoms of uræmia. He lay unconscious, with stertorous breathing, and occasional twitching of the right side, face, and arm. With the right hand he continually scratched the chest and abdomen. The left side seemed to be paralyzed. There was rotation of both eyes, and slight turning of the head to the right. The face was not engorged. The pulse had become slower and full. The legs were not more swollen, but the superficial abdominal veins were more distended than usually. The temperature was subnormal. He died the same day.

The *post-mortem* examination revealed a general infiltration of the right kidney, with encephaloid cancer. The organ was uniformly enlarged to the size of a fœtal head. There was no cancerous growth elsewhere. The vesical plexus of veins contained coagulated blood. A yellowish, firm, partially-adherent coagulum extended up the internal iliacs and the cava to the level of the renal veins. A firm, loose plug was found obstructing the left renal vein. The brain was anæmic and œdematous. There was no marked atheroma, no hemorrhage, no embolus, no thrombosis.

Remarks.—I am sorry that a more careful examination was never made of this patient. His general condition excluded all idea of abdominal cancer, though the persistent swelling of the feet pointed in this direction, together with the hæmaturia. The theory of scurvy seemed to explain all the symptoms, and an examination of the abdomen was neglected, which would certainly have led to the discovery of the tumor.

I thought that an occlusion of the cava and renal veins was the only condition that could give rise to the symptoms present before death, especially after taking into consideration the injection of Monsel into the bladder. Of course I expected to find that the function of both kidneys had been interfered with by the clot. I also expected to find some obstruction in the cerebral vessels.

In the discussion on the diagnosis of renal enlargement, Dr. Guitéras said that his own and the other cases presented should be used as evidences, not of the impossibility of detecting renal enlargement, but of our neglect to look for it. He was sure that in the great majority of these cases the region of the kidneys had not been examined. He remembered a case where Dr. Pepper had diagnosed, by physical exploration, a nephritic abscess, in a case where the symptoms could be well accounted for by an empyema that was also present on the same side. He thought that palpation gave valuable information as to the size of the kidney. He had

found that by frequent percussion he could always satisfactorily map out the organ. Very often in the first examination no results could be obtained, but subsequently one became able to recognize the changes in the conditions of collapse and inflation of the surrounding guts that act as obstacles to the examination. *June 13th*, 1878.

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b. FEMALE ORGANS OF GENERATION.

4. *Fibrous tumors of the uterus—calcareous tumor of the uterus—polypi of the uterus—encysted dropsy of the peritoneum.*

By Dr. H. Lenox Hodge.

This specimen is unusually interesting on account of the varieties of uterine tumors which it presents, and because of the great rarity of cases of encysted dropsy of the peritoneum.

It was removed from a woman about 50 years of age. She had never married. Her menses had ceased about four years ago.

The tumor was first discovered by her family physician some nine years ago, in the upper part of the right inguinal region. There had been a slow increase until within the last four or five months, when the increase became rapid. Early in July last, ascites was first detected: the fluid increased so rapidly, that on the 31st of July she was tapped, and eleven pints of a thin straw-colored fluid were removed. The fluid contained a large quantity of albumen. The fluid accumulated rapidly, and again on the 29th of August she was tapped, and sixteen pints of fluid, similar to the other, were removed. The fluid formed again, and she died from exhaustion September 22d.

A *post-mortem* examination was made forty hours after death. About eighteen pints of straw-colored fluid were found in the cavity of the peritoneum. The peritoneum was everywhere very much thickened, and studded with little prominences, apparently the result of inflammatory action. Strong bands extended from the liver downward and to the right as far as the ilium, and completely separated this portion of the peritoneal cavity from the rest. Even when the fluid was removed from the space below these bands, it remained without leakage in the space above them, and constituted a complete encysted dropsy of the peritoneum.

These portions of the liver, spleen, large and small intestines, and uterus have been brought to exhibit the condition of the peritoneum above described.

The uterus with its tumors so completely filled the cavity of the pelvis, and was so universally adherent, that it could only be removed by stripping the peritoneum from the abdominal walls and pelvis, and then taking away all the pelvic viscera in mass, and the parietal peritoneum with them.

The uterus was exceedingly enlarged, and everywhere in its walls were fibrous tumors, varying in size from that of a marble to that of an orange, Some of them were sub-peritoneal, some in the midst of the walls, and some of them sub-mucous. Many of these tumors had undergone fatty degeneration. One large one had been reduced almost to fluid contents, and in the interior of the cyst thus formed there was a calcareous tumor attached to the wall, about the size of a walnut.

The cavity of the uterus was much enlarged, and contained within it two large polypi. Each of these was about nine inches in circumference and three inches long, and attached to the uterine wall by a narrow pedicle. The lower of these polypi was narrow at its lower extremity, and reached to the internal os-uteri. It completely prevented the passage of a probe, even when the attempt was made after the uterus had been removed from the body, and placed upon a table.

The following memoranda are the results of a microscopical examination made by my friend Mr. B. Mosquera:

Portion from walls of uterus.

1. Spindle-shaped, connective-tissue cells, with a granular protoplasm.
2. Small round granular cells.
3. Bands of wavy fibrillar connective tissue, intermingled with connective-tissue corpuscles.

Contents of cyst formed by degenerated fibrous tumor.

1. Granular matter.
2. Small round granular cells.
3. Oil drops.
4. Débris of spindle-shaped cells.
5. Fatty infiltration and fatty degeneration of connective tissue.

Surface of polypus.

1. Numerous round granular cells arranged in groups.
2. Large number of oil cells.
3. Fatty degeneration of villi.

September 27th, 1877.

Report of the Committee on Morbid Growths:

"A microscopical examination of the uterine new formations presented by Dr. Hodge, shows them to consist of firm, dense fibrillar connective tissue, arranged in bundles running in every direction. The blood-vessels have their walls greatly thickened by an increase of fibrous tissue, and they are few in number. This structure demonstrates the growths to belong to the fibromata.

"From a macroscopical examination, some of the growths are seen to have undergone a calcareous infiltration, others a cystic degeneration. The nodules developed in the peritoneum are the result of an inflammatory process." *October 25th*, 1877.

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5. *Delivery of a large fibroid tumor from uterine cavity.*

By Dr. Michael O'Hara.

Saw Mrs. De C., æt. 32, first on October 28th, 1877, suffering from uterine hemorrhage. She was quite blanched, and in an exhausted condition, from repeated hemorrhages for seven years, as well as from the privation of the necessaries of life. The uterus appeared to be about the size of a five-months' pregnancy, larger and thicker at the left superior portion than at the right side. She declined examination, and was ordered ergot and ol. erigerontis Canadensis, with iron, quinine, and liberal nourishment.

She has no family history of fibroid growths. She menstruated first at thirteen years of age. Menstruation has always been copious and regular. Between her menstrual periods for the last seven years, she has had on an average four additional losses of blood. She married seven years ago, but has never conceived; and her hemorrhages commenced immediately after her marriage.

On 30th, upon examination found os open about the size of a quarter of a dollar, some bleeding, and tumor presenting; no pains. Ergot was given freely. At 11 P.M. found her in active labor, os out of reach, and not to be found, by reason of a large mass blocking up completely the vagina. Dr. Garretson was called in consultation. By dint of use of fingers, inserted into texture of tumor, the application of obstetrical forceps, and the use of the hook attached to the obstetrical forceps, the mass was brought away. Whisky was freely used, but the patient was not anæsthetized, on account of using the bearing-down efforts of the patient, which, with manual compression of the uterus aiding her pains, accom-

plished much. In a short time, and in the same way, the second mass was delivered. After this, by use of the fingers, etc., and with force, all that could be extracted of that part attached to the uterus itself was torn away. Some pieces were left attached to the uterus, on account of the exhausted condition of the patient, to be allowed to slough away if not expelled by the uterus itself.

The mass, though appearing to be composed of three pieces, was really all one tumor, weighing in all about twenty-nine ounces. The first part extruded was really a portion only, and you could see the place of attachment to the second mass, while the pedicle, or rather base, to which the second portion was attached, was quite large, and more fibrous in structure than the tumor, which appears to be undergoing fatty transformation.

The tumor is now much shrunken. One of the masses is oval in shape, four inches long, three and a half inches wide, two inches thick. This was first removed, tearing it from its pedicle of attachment to its fellow, which was one line thick, two inches wide. This mass is very firm, white, and somewhat lobulated.

The second mass was much softer, of the size and shape of a large orange, and was apparently attached to the whole upper portion of the uterus by a broad expanded base of dense fibrous tissue. Three portions of this base, two or more inches in diameter, were each separately removed by force. There was little hemorrhage during the operation or afterwards. There was much exhaustion from pains and the severe contractions of the womb. On ergot, iron, and quinine, nourishment and stimulus, though excessively reduced, the patient is doing well. *November 8th*, 1877.

Report of the Committee on Morbid Growths:

"The tumors removed from the uterus, presented by Dr. O'Hara, are seen to have, upon microscopic examination, the structural elements of fibromata, consisting of bundles of fibrillar connective tissue, arranged without any regularity as to direction, so that a thin section exhibits transverse, longitudinal, and diagonal cuts of the fibres. The cells are not numerous, spindle-shaped and stellate in form, some containing an oval nucleus. The blood-vessels are few in number, and have their walls greatly thickened by connective tissue." *November 22d*, 1877.

6. *Uterine growths.*

Presented by Dr. J. E. Eskridge.

For the specimen and following history of the case I am indebted to Dr. P. B. Breinig, of Bethlehem, Pennsylvania, who writes, "The patient, a lady 45 years old, living in Berks County, Pennsylvania, noticed, about ten years ago, that her menses were very free, and that five years later the discharge was so great as to constitute menorrhagia. Two years ago she observed a hard tumor forming in the left side, low down, since which time more or less metrorrhagia, with contractile pains, has existed. By measurement with the sound, the uterus is found to be five and a quarter inches. There was found within the body of the uterus a roughness, which bled profusely when touched by the sound. The operation of scraping the cavity was performed, and about a tablespoonful of tissue, such as the specimen sent, was removed. There was but little hemorrhage: liq. ferri subsulph. was applied, and when I left the patient rested comfortably. Upon the left side of the uterus there is a fibroid tumor the size of half a fœtal head. There is no apparent cancerous cachexia."

January 24*th*, 1878.

Report of the Committee on Morbid Growths:

"The specimen presented by Dr. Eskridge, and referred for examination to the Committee on Morbid Growths, is seen to consist of numerous cysts lined with epithelial cells, and of bands of fibrillar connective tissue covered with epithelial cells, forming papillæ. The intercellular fibrous tissue between the cysts, and that forming the papillæ, is permeated by numerous dilated capillary blood-vessels, which at some places have ruptured, causing an extravasation of blood between the fibres. There is also an infiltration of large oval or round granular cells, containing large nuclei, in which are seen two or three large nucleoli. Other cells, somewhat smaller, spindle-shaped, and nucleated, are found, with many embryonic cells. The specimen from which the examination was made being only a scraping from the mucous membrane of the uterus, it was not possible to ascertain with certainty the exact nature of the new formation; but from the arrangement and structure, as above described, we are inclined to consider it a cystic papillary adenoma."

February 14*th*, 1878.

7. *Fibroid polypus of the uterus.*

Presented by Dr. Frederick P. Henry.

Mrs. K., æt. 48, first came under my observation at the Episcopal Hospital in January, 1877. Her only complaint was of pain in the back. She was questioned regarding uterine symptoms, but her answers were such as to lead to the opinion that the uterus and its appendages were in a normal condition: at any rate, she was not subjected to a uterine examination. The woman's appearance indicated an excellent state of health. Very shortly after her admission she was attacked with rheumatoid arthritis of the finger-joints, which confirmed the opinion that the pain in the back was of a rheumatic nature. The rheumatoid arthritis ran a rather acute course for that disease, and at its termination she was discharged at her own request, the pain in the back being somewhat relieved.

I did not see the woman again until September last. She did not come to me then for treatment, but mentioned incidentally that her backache continued, and was rather worse; she was at that time under the treatment of another physician. I did not see her again until March 13th, when she came and told me she had decided to place herself under my care. I advised a uterine examination, as on questioning her closely I found that, there had been almost constant bloody discharges from the uterus for nearly two years. She preferred first trying medical treatment. In about ten days, the medicine proving of no avail, she agreed to the examination. I discovered a tumor presenting in the cervix, and advised its immediate removal. This was on Friday, March 22d. On the following Monday, with the assistance of Dr. Nancrede, I removed the growth with the wire écraseur. There was some slight hemorrhage, which was controlled by the application of Monsel's solution. The operation was performed without an anæsthetic. The woman's condition is now wonderfully improved. All uterine discharge has ceased. The pain in the back has gone, and food, formerly loathed, is now taken with a vigorous appetite.

The removal of these growths is not solely indicated by the symptoms, which are not always so distressing as in the present case, but also by the possibility of a malignant sarcomatous degeneration occurring in them. In this degeneration the pedicle is often the last portion attacked, so that removal of sarcomatous polypi is frequently followed by a radical cure.

March 28th, 1878.

c. MALE ORGANS OF GENERATION.

8. *Epithelioma of glans penis.*

By Dr. Comegys Paul.

On the 15th of last May I first saw the patient who furnished this specimen. The glans penis was then a mass of ulceration, with exuberant granulations and many minute sinuses, from which a very slight pressure would cause to appear numerous vermicular bodies of a caseous consistence.

Nine months before, he first noticed a small ulcer situated in the depression behind the corona glandis, and involving the skin only. It gradually increased, although skilfully treated by his medical attendant. When he came under my professional care, I made efforts to cut down the granulations by caustics and stimulating ointments, and laid open the sinuses above mentioned, besides giving attention to his general health. He is a married German, aged 52 years, and has never had syphilis. It was at first suspected that his condition had a syphilitic origin, and specific treatment was at one time adopted, but without any benefit, as was also the effect of all other treatment. Loss of sleep at night from pain, and the long-continued discomfort, at length began to undermine his strength, and led me to recommend amputation. This he readily agreed to, and the operation was performed two weeks ago with entire relief. The method chosen was that of Ricord, in which, after the division of the parts by a circular sweep of the knife, the urethra should be split at several points and united to the skin with interrupted sutures. The result in this case is a stump about two inches long, the end presenting all the characteristics of a healthy wound, and partially covered with a foreskin.

September 10*th*, 1877.

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9. *Cystic sarcocele of testis.*

Presented by Dr. Comegys Paul.

This specimen was removed from No. 8684, a convict incendiary at the Eastern Penitentiary. He is 34 years old, of a scrofulous tendency, but enjoys good health. Fourteen years ago he had an attack of gonorrhœa, during which he slipped and strained himself violently while playing a game of ten-pins. Swelling and pain in the left testicle followed, with which he was confined to his bed for a week. The testicle has never been

in healthy condition since, and it has received several injuries from kicks, blows, etc., in the mean time. It has been enlarged and growing slowly during the whole of this period, but has never occasioned much pain, until the last fifteen weeks, when it assumed a dull aching character, which extended to the lumbar region. Because of this excessive discomfort I removed it, with the assistance of Dr. E. C. Hines, on the 7th instant.

He has been in good and comfortable condition since the operation. Two ligatures were required, neither of which has as yet come away.

March 14*th*, 1878.

VI.—THE NERVOUS SYSTEM.

1. *Spina bifida.*

Presented by Dr. William H. Parish.

M. R., a female child, came under my observation, when two weeks of age, at the clinic of diseases of woman and children at the Jefferson College Hospital. She was of small size, very feeble, and extremely restless.

A spina bifida existed over the lower lumbar and the sacral region. The tumor existed at birth, and, when first seen by myself, was about the size of a small orange, tense, slightly fluctuating, with a glistening, reddened, somewhat umbilicated surface.

The lower extremities were disproportionately small, with evidence of imperfect vascular circulation. They were quite motionless, and there was marked double talipes equinovarus. There were frequent small fecal evacuations, and frequent or continuous urination.

The extreme debility of the child, the apparent absence of control of the bladder and rectum, and the complication of aggravated club-foot, seemed to contra-indicate any operative interference with the tumor.

The only local treatment was that of gently supporting the mass, by means of a pad of cotton and a bandage.

Later, superficial excoriation occurred, and a few days before death there was a spontaneous escape of cerebro-spinal fluid, followed in a short time by an escape of pus. The mass diminished in size, and the child became feverish and more restless. Death, preceded for a few hours by convulsions, occurred seven weeks after birth.

We are indebted to Dr. P. E. Loder for the removal of the specimen.

This, as presented, consists of the lower four lumbar vertebræ and the two sacral vertebræ. Only the two lower lumbar and the sacral vertebræ are defective. From the appearance of the two upper sacral vertebræ it seems probable that the rest of the sacral canal was also incomplete. The deficiency is due to the non-approximation of imperfectly-developed laminæ, and the absence of the spinous processes. The tumor is continuous, by means of a constricted portion, with the contents of the spinal canal. It consists of the arachnoid and dura mater, with the overlying integument. The cord itself is continuous into the tumor, and terminates by union with

the integumentary covering. A circular area, of a color deeper than that of the surrounding skin, marks the spot of union between the cord and the integument. The sac contained a small amount of pus. Through the upper anterior sacral foramina can be seen passing the first anterior sacral nerves. *November 8th*, 1877.

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2. *Laceration and inflammatory changes in the spinal cord resulting from fracture of the cervical vertebræ.*

Presented by Dr. J. Solis Cohen.

The case from which the specimen was obtained was under the care of my colleague, Dr. Marcus Franklin, in the surgical wards of the German Hospital.

H. P., æt. 24, was admitted to the German Hospital, August 22d, on account of an injury received in falling backwards from a window fourteen feet from the ground, and striking the back of his neck upon some loose bricks. On admission there was complete sensory and motor paralysis, extending from the sixth cervical vertebra downwards. The muscles responded to the induced current. The fæces were passed involuntarily, but there was retention of urine, and he was unconscious of the introduction of the catheter. The respiration was entirely diaphragmatic. His appetite was good, and he expressed himself as feeling first-rate. In a few days excessive tympanitis supervened, with constipation, which was only partly relieved by active cathartics and enemata. Wet cups, followed by ice, were applied to the spine.

The temperature in the mouth gradually rose to 103° F., but the extremities were cold. On the third day he complained of something in his throat (a sensation apparently due to paralysis of the pharynx), and he had some difficulty in deglutition. Articulation was somewhat impaired. In other respects he felt pretty well, talked cheerfully, and had no idea of his critical condition. On the fifth day a partial and very incomplete sensation returned to the upper extremities, but the paralysis of motion continued complete. Some delirium was now observed, which was followed by epileptiform convulsive movements, which were not well marked on account of the paralysis. These symptoms increased till the time of death, which occurred on the tenth day after admission.

The immediate cause of death appeared to be due to the interference with the respiration, owing to the paralysis of the scaleni and intercostals, and no doubt increased by the pressure of flatus on the diaphragm. At

the *post-mortem* the fourth and fifth cervical vertebræ were found to be fractured through the laminæ, and the spinous processes to be driven in upon the cord, which was quite disorganized at this point. The softening extended up a short distance. There were also evidences of miningitis.

Examination of spinal cord after immersion in alcohol during four months. By Dr. Morris Longstreth.

At the seat of injury, the dura mater, for the length of a finger's breadth, hangs like a loose sac; for the length of three-fourths inch above and below, the spinal cord is enlarged, and the dura over it is expanded and pretty tensely stretched. On laying open the dura mater, it is found thickened, especially on its posterior aspect, some distance above and below the seat of injury, especially in the latter direction. The arachnoid membrane is pretty completely adherent to the dura, in the length of two and a half inches on the posterior surface, whilst anteriorly the adhesions are less general, and not so firm, so that the cavity of the arachnoid was closed at this part; there is no evidence of hemorrhage into it. In the subarachnoid space, below the seat of injury, are several blood-clots, losing their color and undergoing absorption. At the seat of injury the arachnoid and pia mater are fused together, and constitute a thick wall to what is now a cavity in the centre of the spinal cord. On dividing the membranes at this point, all semblance of the cord has disappeared; a little semi-fluid nervous matter is seen partially filling the space; from above and below the much-softened nervous tissue bulges inward, and forms the roof and floor of the cavity of an abscess. The softening of the cord extends both above and below the seat of injury for some distance, gradually be coming less until the cord and membranes assume a normal consistence and appearance.

The microscope shows the ordinary appearance seen in nervous tissue resulting from contusion, and the subsequent inflammation and softening, —fragments of tubules, numerous small nuclei, granules and oil globules, blood-coloring matter and large granular corpuscles.

Dr. Shakespeare remarked, that the specimen showed a complete destruction of the nervous structure of the spinal cord at the place of injury.

He thought the statement that "there was a partial return of sensation" in the upper members misleading. It was based upon no other fact than "the existence of pains," which might have been entirely subjective, since the sensibility of the upper members was not tested by pinching, pricking, or by any other means. The same sort of evidence could be made to prove a return of sensation in an amputated limb when irritation of the nerve-

stumps produced a sensation, which the patient referred to the ends of his absent fingers.

The so-called "return of sensation" in the case reported can be very readily understood, if one conceives an irritation at the point of injury, either inflammatory or by spiculæ of bone,—the reporter states that these were found in the mass of detritus between the interrupted ends of the cord,—or by any other means whatever, of the central ends of the sensitive fibres from the arms as they pass up the columns of the cord, thus causing a sensation which, although it would have its origin at the point of injury, yet would be referred by the patient to the peripheral termination of those fibres. *January 10th*, 1878.

VII.—ORGANS OF SPECIAL SENSE.

1. *Bean removed from external auditory meatus.*

By Dr. Carl Seiler.

Maggie E., æt. 12, applied to me for relief. She complained of deafness and pain in the right ear. On questioning, she and her attendant said that about eighteen months ago her hearing had become bad, and that the pain had only been felt for the last four months. On examination I saw a hard white body filling the entire meatus externus with the exception of a small slit-like opening near the top of the inner wall.

Before proceeding to remove the obstruction I questioned the patient closely as to the time when she had put the foreign body into her ear. She, however, denied all knowledge of having put anything into her ear at any time.

In trying to remove what afterwards proved to be a bean, I found the forceps of no avail, and had to resort to a hook the point of which was strongly curved while the body of the instrument was but slightly bent. With this instrument, which was made at the time out of a hair-pin, I was enabled to pass through the narrow opening and behind the bean, and then by pulling upon the hook a very little force sufficed to dislodge it.

After the removal of the bean, the canal was found to be but partially filled with dry cerumen and epithelial scales, while the tympanum presented the bright-red appearance of a tympanitis. *October* 11*th*, 1877.

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2. *The growth of the fungus aspergillus in the human ear.*

By Dr. Charles H. Burnett.

The aspergillei belong to the division of the fungi called arthrospores. In the same divison are other important parasitic fungi, viz., the tricophyton, the microsporon, the achorion, and the oïdium.

The aspergillei form a family by themselves, according to Léveillé, and under the genus aspergillus Charles Robin enumerates eight species. Some of these species have been found in tuberculous lung, notably in

the lungs of birds; but the most interesting species are those which grow in the auditory canal of man.

The fungus aspergillus is not uncommonly found growing in the human ear, where it produces an inflammation of rather a peculiar form. This inflammation is, however, frequently assigned to other causes, and is very often mistaken for simple eczema, which it may accompany or excite.

Doubtless the aspergillus grows often in the ear without being recognized. Hinton, of London, with his enormous field of observation, states that he had not met it, and doubted its existence in denizens of cities. Dr. J. P. Cassells, of Scotland, is the only British writer on the observation of this peculiar aural fungus. Blake, of Boston, thinks it would be much more frequently detected if looked for. It is without doubt often washed from the ear in large masses, and termed wax, by superficial observers. Wreden, of St. Petersburg, who has reported more cases than any other observer,—between sixty and seventy at the last report,—calls the aspergillus the *specific aural fungus*. He claims to have found only two varieties in the ear, the *yellow* and the *black*. The latter is very much more common in its occurrence than the former. Some writers consider the aspergillus found in the ear as modified and rendered peculiar by its growth in the ear. Hallier, of Jena, considers aspergillus very similar to penicillium or mould, but that the latter is a larger fungus. One fact is fully established: the aspergillus shows a more marked tendency to grow in the human ear than any other fungus.

The symptoms of its growth in the ear, and the treatment to be pursued, may be learned from the account of the following cases and specimens:

Specimen No. 1. *Aspergillus nigricans.*—This specimen of aspergillus was removed from the ear of a young merchant, 28 years old, while on a visit to the Centennial Exhibition, September, 1876. The patient stated that he had felt some slight discomfort, itching, and pain in his left ear several days before he came under my notice. The pain in this case gradually increased, until he was obliged to relinquish all efforts at seeing the Exhibition, and seek medical aid.

On examining the ear in this case, the external auditory canal was found nearly entirely filled with the black mass I here show you. Upon subjecting small portions of this mass to the microscope, the peculiar nature of the offending body was revealed. The entire mass was not removed at the first sitting, as the inner surface of the fungous growth was adherent to the membrana tympani. But after the use of alcohol instillations for twenty-four hours, the masses here exhibited were removed, placed in pure glycerin, and have there remained unchanged until now.

After the fungus was entirely removed from the ear, the meatus and the drum-head were seen to be inflamed; but under the use of diluted alcohol, all remaining spores of aspergillus were destroyed, and the various portions of the ear assumed their normal appearances and functions.

Specimen No. 2. *Aspergillus nigricans.*—This small specimen of aspergillus nigricans is interesting chiefly on account of a very pertinacious regrowth. The quantity exhibited this evening is small, but represents the size of five or six masses removed successively from the same ear in the course of a week or ten days.

The patient, a wine-merchant of this city, was 67 years of age, strong and well. He could give no explanation as to the probable cause of growth of this parasite in his ear. He had first noted, some weeks before I saw him, a buzzing in his left ear, accompanied by a diminution of hearing and a slight itching pain. A very slight, *watery* discharge had run from his ear at times since these symptoms were felt. His physician had syringed the ear several times, and had washed out each time a substance, said to be cerumen; but on account of the recurrence of all of the above-named symptoms the case was deemed worthy of a change of treatment.

There was then detected, deep in the anterior part of the auditory canal, near the membrana tympani, in the angle formed by the latter and the anterior wall of the external auditory canal, the dirty-white mass herewith shown you. Its true nature was discovered by a microscopic examination.

After a thorough cleansing of the ear, instillations of alcohol were applied to the meatus once daily, and the ear syringed regularly thrice daily, whereupon the symptoms of disease abated. With an attack of small furuncles in the canal, there was a recurrence of the aspergillus; but all traces of this annoying parasite, at last, in the course of ten days, disappeared under the use of diluted alcohol (water and alcohol equal parts) and warm water syringing thrice daily. The ear then resumed all its normal functions. This man was the oldest person in whose ear I have found the aspergillus growing. The fungus is not found in the ears of children. It is most likely to affect adults between twenty and forty years of age.

Specimen No. 3. *Aspergillus nigricans.*—This large mass of aspergillus nigricans, mycelium, etc., was removed from the left ear of a large, strong Englishman, 35 years old, a wool-carder in one of the mills in the northeastern part of our city. In this instance there was excited by the presence of the aspergillus in the ear, considerable eczema and some myringitis,—*i.e.*, some inflammation in the outer layer of the drum-head. The case was persistent, as all cases of aspergillus growth are, when accompanied by eczema of the external ear.

The treatment pursued in this case at first was alcohol undiluted, but this proved too stimulating to the eczematous inflammation, and consequently dilute alcohol was substituted, and the syringing of the ear carefully pursued, in order to remove the fungus and its spores.

In this case there were two diseases,—the one, the eczema, dependent on the other, the growth of a vegetable parasite on the skin of the external auditory canal and drum-head. The treatment adapted to destroy the fungus was likely to increase the eczema, and doubtless did make it worse in this case for a time; but as the eczematous disease in such a case is dependent upon the presence of the fungus, the surgeon is justified in adopting and continuing a treatment most likely to destroy the parasite.

Respecting the use of alcohol instillations in the treatment of aspergillus in the ear, it must be borne in mind that all ears do not tolerate the undiluted alcohol, especially if the skin is at all broken. It is safer, therefore, to begin with instillations of equal parts of alcohol and water.

Specimen 4. *Aspergillus flavescens removed from the ear of a man some years ago.*—The fungus is seen imbedded in fatty matter,—*i.e.*, in oil put into the ear by the patient to relieve the annoyance caused by the growth of the parasite. The chief interest in this case is the opportunity it offers to compare the shape of the sporangia of this variety of aspergillus with those of the other three specimens. It will be seen that the fruit-stalk and head in this case are smaller than in the black variety, and that the sterigmata are wanting around the lower part of the receptaculum. I am not aware that the growth of this variety of aspergillus excites symptoms different from those caused by the growth of the black kind in the ear. (Fig. 3, A.)

The specimens I present this evening illustrate the more common varieties of the aspergillus. The macroscopic appearances of a mass of this fungus, as found in or washed from the ear, are worthy of attention. For although the microscope must in every case decide the presence of aspergillus in a suspicious object coming from the external auditory canal, nevertheless the macroscopic features of such a mass are of such a nature as to lead the surgeon to suspect that he has to deal with a case of aspergillus in the ear. If an ear containing a mass of aspergillus be examined by means of an ear-mirror and ear-funnel, it will present most usually an appearance which will lead to the supposition that the ear is occluded not by wax, but by a foreign matter, which has life, and is hence more or less irritant. Lifeless foreign bodies do no harm to the ear if left alone, but a living foreign object, either animal or vegetable, will invariably excite inflammation sooner or later. If the fungus has not been growing long in

the ear, merely a patch of pale yellow pollen-like matter will be detected at the fundus of the auditory canal. This small colony of spores just developing into filaments, for such it is, is usually situate on the membrana tympani, or very near it. In any case, whether the first deposition of spores occurs there or not, the tendency of the aspergillus is to grow over the drumhead first, and from that point it spreads outward, covering the wall of the meatus, until a hollow cast of the canal is formed by the vegetable parasite. This pollen-like appearance is seen only in the very earliest stage of a growth of that which is finally a so-called lardaceous-looking or false membrane, either partially or entirely filling the external auditory canal. If the ear has been invaded by either variety of aspergillus, the growing mass has a yellowish or dirty-white appearance. It looks, in fact, like a piece of sodden newspaper, especially if the fungus is old, when it assumes a darkish-brown color in some cases. Sometimes the ear may seem to be plugged with a black substance looking not unlike wool. An inexperienced eye might conclude that the occluding plug thus formed is of hardened ear-wax; but ear-wax is never literally black, nor does it excite the other symptoms of the presence of aspergillus in the ear, viz., pain and inflammation.

A mass of aspergillus does not lose its coherence when subjected to immersion in water or glycerin, but a lump of ordinary hardened cerumen soon melts, and is diffused throughout the water or any other fluid in which it is allowed to lie. This coherence on the part of a mass of aspergillus would distinguish it from one of ear-wax. The microscope would immediately show whether such a mass washed from the ear were aspergillus or the peculiar laminated epithelial plug, the so-called *keratosis obturans* of Wreden.

As a matter of fact, therefore, we do find whitish and decidedly black masses of aspergillus in the human ear, and not uncommonly there are found masses which are in the main whitish, but which are thickly studded with black spots or are as it were stratified with black.

I may say, in passing, that it is claimed by several authorities in Europe, and by some in this country, that varieties of fungi not aspergillus have been found in the human ear; but these are of infrequent occurrence, and would, by their bluish or red color, attract attention and at once decide their nature as not that of ordinary aspergillus. There is, too, a rare form of aspergillus, known as the ascomycete form, *i.e.*, a form which has flourished so completely as to reach the condition of bearing what is known among mycologists as utricular fruit. This form is said by Wreden to have been found only once by him, and to have been of a beautiful dark

red color. It is of so rare an occurrence as not to enter into our consideration at present. Dr. J. Orne Green,* of Boston, has described a form of aspergillus rubens, in which the fungus was, as indicated by the name given it by Dr. Green, of a red color.

The opportunity of observing the early stages of growth in a colony of aspergillus is of course rare. It can only offer itself where the ear is being examined or treated for some other complaint, since the early stages of growth of aspergillus in the ear excite no subjective symptoms which would lead the patient to seek medical aid. As a rule, in cases of aspergillus the patient seeks relief for hardness of hearing, which has come upon him rather suddenly, and has been attended by a pricking sensation, —sometimes by considerable pain. The pricking or pain has been followed usually by a slight watery discharge, after which the pain has perhaps ceased, but the hardness of hearing continues. Sometimes the pain attending the growth of aspergillus in the ear is very severe, as it was in the case which furnished the remarkably large, deep black mass I have shown you this evening, in specimen No. 1.

When upon inspection, such a mass is found in the external auditory canal, the first impulse is, or should be, to syringe the ear with warm water. If spontaneous detachment has occurred, the offending matter can usually be syringed out with ease. In most cases parts of it at least, can be washed out, and furnish, on examination, a more accurate clue to the true nature of the mass, than the mere inspection with the ear-mirror. The aspergillus tends, however, to root itself upon the membrana tympani, —to seek, in fact, the most secluded parts of the ear; and it has been known to pass into the tympanic cavity after having destroyed the membrana tympani, as in a case reported by Politzer. But this is a rare occurrence. So far as my observation goes (extending, as it does, over some twenty cases), spontaneous detachment of the false membrane, from the membrana tympani, is the tendency in all cases of aspergillus in the ear. Where this does not occur, it must be due to the fact that the membrana tympani is abnormally thin and yields to the grasp of the rootlets of the fungus, thus favoring a tighter hold, or there must have been a cicatrized or partially closed perforation in the drum-head which favors the grip of the aspergillus and aids its entrance into the drum-cavity. I have observed one case of aspergillus growing *in the tympanic cavity*,† though I strongly suspect that the case furnishing the specimen of aspergillus shown in

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* Proceedings of Boston Society of Medical Sciences, 1875.

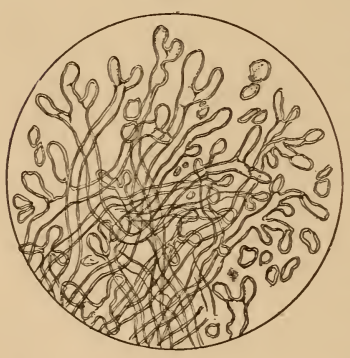
† Transactions International Otological Congress, 1876.

Fig. 2 was one in which some of the fungus had invaded the tympanic cavity.

In most cases syringing will remove the major part of the fungous mass at one sitting, but, of course, enough spores will probably be left behind to engender a fresh growth by the next day. Hence it not uncommonly happens, that an ear which has been entirely cleansed, so far as the eye could detect, of what was supposed to be cerumen, is, to the great surprise of the surgeon, filled in a day or two with a plug as large as the one previously washed out. In such cases it will be found that the fresh growth springs from the angle formed by the anterior wall of the auditory canal and the anterior half of the membrana tympani. Here, spores having become deeply rooted, at least continued syringing must be practised to effect their destruction and removal. Both their destruction and removal must be hastened by the instillation of some of the numerous parasiticides. All things considered, I would give the preference to alcohol, either undiluted or in various proportions with water. In one case it seemed that the cure was hastened by insufflation of a powder, composed of equal parts of magnesia and salicylic acid, into the auditory canal, after all macroscopic portions of the fungus had been removed by means of the syringe.

Microscopic features.—The microscopic features of the growth of this parasite are varied and full of interest. If a small piece of a colony of aspergillus in the earliest stages of development be examined under the microscope with a power of about 250 diameters, a picture similar to that in Fig. 1 will be observed. It is in fact the first formation of rootlets or the mycelial web, from which later the fruit-stalks spring. It will be seen that some of the filaments composing this web tend to become bulbous at one end, and that the latter, as the stem grows, becomes larger and dotted, until finally there is standing out from the dense web of mycelial filaments a perfect fruit-stalk and a fructiferous head, studded with short peg-like limbs on the free ends of which are the spores.

Fig. 1.



The passage of the bulbous stem to the more perfect fructiferous hyphen or fruit-stalk, as these parts are termed by mycologists, can be seen by referring to Fig. 2. All of these stages I have traced in specimens of the fungus removed from the human ear. In the fluid parts of the specimen,

epithelium in small quantities may usually be seen, as in the upper left hand corner of Fig. 2.

Fig. 2.

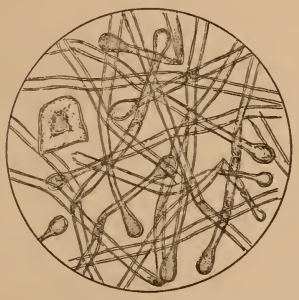


Fig. 3.

Very rapidly the perfect fruit-stalk is found in large numbers, and in all stages of development, and the mycelial rootlets will be seen to be coarse and septate. All of these later features may be seen in the same microscopic specimen. On one hand may be seen a well-formed though unripe hyphen or fruit-stalk, while in the centre of the field or on the other hand may be seen the ripe aerial fruit, from which the fully ripe spores drop literally in myriads. (Fig. 3, C.) The latter rapidly sprout and carry on the processes of development of their luxuriant progenitors.

The characteristic microscopic difference between the two varieties, *yellow* and *black*, is seen in the shape and size of the two parts, the *receptaculum* and *sterigmata*, forming the so-called "head."

In the *Aspergillus nigricans* (Fig. 3, B), the sporangia or heads are distinguished from those of the *A. flavescens* (Fig. 3, A) by the fact that in the first the sterigmata cover the receptaculum on all sides, while in the latter form the lower fifth or fourth of the receptaculum is entirely free from sporangia.*

So far as the size is concerned, it may be said that generally the head in the yellow variety is smaller than in the black aspergillus. The color is not a safe point of differential diagnosis; the names so far as they are based on coloring are almost arbitrary. The color of the various portions of any aspergillus may vary from palest yellow to a blackish or yellowish brown. The microscopic examination of a specimen of aspergillus is aided by subjecting the latter to a drop or two of liq. potassæ.

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* Wreden, Archives of Ophthalmology and Otology, 1874.

To sum up, then, it may be said that in general there are found four distinct elements in specimens of aspergillus taken from the human ear:

1. The mycelial web, composed chiefly of the so-called tubules, filaments, or rootlets.
2. The fruit-stalk or hyphen, and
3. Its head.
4. The free spores.

The first, the filaments or tubular rootlets, are composed of sections, and are hence described as septate. The component cellules are from 0.090 mm. to 0.020 mm. in length.

The fertile hyphens or fruit-stalks vary in diameter, the latter being from 0.009 mm. to 0.013 mm.; their length is about 0.770 mm. These terminate in the so-called *receptaculum*, the placenta of Micheli, which has a diameter of 0.028 mm. (Robin).

On this grow the sterigmata and spores, which, together with the receptacle, form the so-called capital or head, 0.060 mm. in diameter, the third of the elements named above.

The spores are spherical, and measure 0.003 mm. in diameter. By careful fine adjustment of the lens, their surface is seen to be echinate. Some idea of the immense number of these spores in any case may be gained by a knowledge that Pacini, quoted by Robin, estimated that there are nineteen thousand spores on each capital or fruit-head.

Etiology.—It has usually been supposed that the dampness and squalor of the dwellings of the poor have been the fruitful causes of the growth of aspergillus in the ear. My experience has led me to a somewhat opposite conclusion, since I have observed more cases of aspergillus in the ears of those living in good hygienic surroundings, or even in luxury, than in the poorer classes. It appears, indeed, that aspergillus will grow in any ear which has been the seat of a morbid discharge of any kind. Even a scratch, as from the nail or a pin, which leaves behind it a minute quantity of blood, becomes a soil favorable to the growth of aspergillus. In fact, any dead animal or vegetable matter, especially if the latter be oleaginous, when left in the external auditory canal, will favor the growth of aspergillus. Hence the popular use of oil for various aural maladies is not uncommonly the direct cause of the growth of aspergillus in the ear.

Therefore, after any aural disease which has been attended with a morbid excretion, has entirely disappeared, the remnants of the hypersecretion should be washed out and the auditory canal left in a clean state. There is no evidence that the aspergillus grows on the natural ceruminous secretion of the auditory canal. It appears, indeed, that but for the presence

of the cerumen in the canal, the ear might be invaded more frequently by the aspergillus, since the latter seeks a secluded spot for growing. One of the protective functions of cerumen is shown, I think, in the fact that in an ear the canal of which is sheathed with normal ear-wax, aspergillus is never found in a flourishing condition, while, in the ear invaded by aspergillus, cerumen is rarely if ever found in either a normal quantity or condition.

Treatment.—The treatment of this annoying disease, caused by the growth of aspergillus in the ear, and termed *Otomycosis aspergillina*, or *Myringomycosis aspergillina*, has already been suggested. It consists chiefly in the entire removal of the fungus and its spores. In this the use of the syringe is the main reliance; but this must be aided by the instillation into the ear of a fluid favorable to the destruction of a vegetable parasite. For the latter purpose rectified spirits of wine, either diluted with water or in full strength, should be employed several times daily, in quantities varying from fifteen to twenty drops. The ear should be carefully examined by means of the ear-mirror and speculum each day, and the treatment modified according to the stages of the disease. The least irritation of the ear, combined with the most effectual mechanical removal of the parasite, will give the most satisfaction to both patient and surgeon. *April 11th*, 1878.

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3. *Mastoid disease—Trephining—Death—Clot in the lateral sinus.*

Presented by Dr. H. S. Schell, for Dr. William W. Keen.

Joseph Juddy, æt. 17, admitted to St. Mary's Hospital December 9th, 1875. When æt. 3, had the measles, and has had discharge from both ears, off and on, ever since. Four weeks ago had fever, and ear has been discharging freely. An incision had been made soon after over left mastoid process, opening an abscess. Has had some symptoms of meningitis.

December 10th.—Had a rigor lasting an hour.

December 11th.—Condition.—Is totally deaf in left ear, and moderately so in the right. Is pale, trembling, sweating. Tongue clear. Left pupil a little larger than right, and reacts to light. A blindness often seizes him on attempting to rise. Pulse 132. Intelligence good, but a little confused. Memory poor. On inserting a probe, dead bone is found on mastoid process, and the probe glides over its surface anteriorly into the external meatus, and also passes up into the squamous portion, one inch above and in front of the auricle, dead bone being felt all the way.

December 12*th.*—*Operation.*—With Drs. Schell and Burnett in consultation, I made an incision back of the auricle, on the anterior edge of the mastoid, the other incision being too far posterior, and, with A. H. Buck's conical trephine, opened the mastoid, the walls of which were as yet intact. The opening was then enlarged with a small bit, and about a drachm of thick, cheesy pus evacuated. No loose bone was found. A tent was inserted, a poultice applied, and the wound ordered to be syringed daily with a solution of gr. ij chloral to f℥j of warm water.

December 14*th.*—Had a rigor of half an hour's duration. Left ear is one and a half inches lower than right. No protrusion of eyeballs; no œdema of lids. Discharge more copious and better.

December 15*th.*—Appetite good; general appearance good; pulse 94. Neck feels stiff, and complains of trapezius and all the back of the neck. Coughing is painful. Ordered tonics and nutritious diet.

December 22*d.*—Has had four rigors within a week, sometimes accompanied with vomiting. Has violent night-sweats. General appearance very bad; face flushed; great and constant headaches. On examination, his eye-ground showed distinct "choked disks," the borders of the papillæ being scarcely at all defined, and its surface almost as red as the retina.

December 23*d.*—Deeming it probable that pus was forming on the inner surface of the necrosed squamous portion of the temporal bone, separating the dura mater from the bone, I called a consultation to decide the question of trephining this portion of the bone. If an opening already existed at the tegmen tympani, no harm would be done by a small and carefully-made opening, and if no previous one existed, and there was pus present, as every indication seemed to show, an opening here at the most likely spot would evacuate it, or, if the pus were over the petrous portion, would open "a path of least resistance," which would be followed by the pus if it was external to the dura mater. If in the brain, no further harm would be done, and the indications might be such as to enable us to relieve the patient by its possible evacuation. After a consultation it was deemed wise to make an exploratory operation.

Having etherized the patient, I made an incision from the old cut over the mastoid, horizontally, just above the ear, down to the bone, and as far forward as the middle third of the zygoma.

Two branches of the temporal were ligated, the necrosed bone was cleared of the overlying soft parts, the ear pulled down, and thus the root of the zygoma exposed. Directly above this, and on a line with the external auditory meatus, I applied a conical-pointed trephine, and carefully

cut through the thin and softened bone. This spot would admit me to the inner surface of the dead bone, and also would avoid in all probability the posterior branch of the middle meningeal.

Having penetrated the bone by an opening about the eighth of an inch in size, I saw the dura mater exposed. With great care I enlarged this by a drill to about one-quarter of an inch in size. The bone was not brittle, but fibrous, soft, and pliable. A small bubble of air escaped from the opening the moment the trephine was withdrawn, but it may have entered from the exterior. No pus at all escaped. With a bent probe I explored the interior, carefully hugging the internal surface of the bone, and applying almost no force. The dura mater was detached all around the opening, and downwards and forwards; the probe easily entered for more than one inch, but no pus escaped. The dura mater was not inflamed, and it did not bulge out, as if pus existed in the interior of the brain; so I did not open it. Having thus established an opening, it was deemed imprudent to proceed further, and the soft parts were partially closed by four stitches, leaving the posterior inferior portion largely open for discharges, and a tent of silk was inserted. He reacted well, and expressed himself as feeling comfortable.

December 24*th.*—Doing excellently. Slept well. Pulse 64, soft and good. Eye-ground as before. Wound looking well, and fearing lest the tent would irritate the dura mater, it was removed. Had a rigor of only half an hour's duration.

December 25*th.*—Wound discharging slightly. Discharge of pus from the old one as before. Slept moderately well. Slight chill this morning, which was apparently cut short by a hypodermic of one-quarter grain of morphia. Head clearer; quite cheerful, and feels better than before the operation. Pulse 70. Ordered quinine.

December 26*th.*—Another slight chill this morning; checked by morphia. Slept only fairly. Mental condition good, but does not seem so well. Appetite poor. No vomiting. Has some headache. Pulse 80. Wound looking well, and discharging as before.

December 28*th.*—Considerable fever. General condition as before. Chills each day. Pulse 120.

January 3*d*, 1876.—Chills daily. Fever at irregular hours. Night-sweats, for which atropia was ordered. Appetite poor. The wound has discharged one or two ounces of fetid pus to-day, by the opening in the mastoid. The rest of the wound is healthy. Pus discharged from among the muscles of the neck, below the mastoid process.

January 12*th.*—Gradually he sank, exhausted, and died to-day. His

mental condition was excellent up to within an hour of his death. Eye-ground as before. No paralysis; no squint; no priapism.

January 13th.—Post-mortem, twenty-two hours after death.—Rigor mortis well marked. Great emaciation. The head only could be examined. The calvarium being removed, the dura mater was not found inflamed over the hemispheres. The cerebellum, at a point corresponding to an opening from the left mastoid, described hereafter, was disintegrated over a surface three-quarters of an inch in diameter, and its borders firmly adherent to the dura mater and the bone. The softening extended about a quarter of an inch into the cerebellar substance, and was pretty sharply defined. A similar point of softening existed on the upper and posterior surface of the cerebellum, beginning at the torcular Herophili, and extending one inch to the left, corresponding to the inferior curved surface of the left lateral sinus. This extended about half an inch into the brain-substance. Save these two points, the brain was absolutely normal. The dura mater at the point where I had trephined was adherent to the scalp, and was not altered in any way. The cerebral arachnoid, at the same spot, showed a barely visible white color over a spot about one-third of an inch in diameter. The cerebral substance was not inflamed. The entire left lateral sinus was plugged up by a suppurating gangrenous thrombus, which extended up to the torcular Herophili, but not to the right of it. The left petrosal sinus was also filled by a thrombus, which was not adherent; was discolorized, but not softened, and only partly filled the sinus. The left inferior petrosal sinus and the jugular vein were not plugged up. The right sinuses were all free. The opening made in the mastoid extended into the cells, and on the inner surface of the groove for the lateral sinus was found a corresponding opening, extending directly into the lateral sinus. Two openings existed in the cerebellar surfaces of the sinus in direct contact with the disintegrated brain-substance, so that a probe could have been pushed from the exterior directly through the mastoid and the sinus into the cerebellum itself.

Below the mastoid, and in the muscles and connective tissue of the neck, was a suppurating sinus. Unfortunately, the examination could not be extended to the heart and lungs. Owing to the excessive cold, I forgot to remove the posterior halves of the eyeballs to examine the disks. The optic nerves were not œdematous, nor inflamed, so far as removed with the brain.

Tympanic cavity.—The tegmen was opened; pus found in the tympanum; ossicles were present and *in situ;* manubrium eroded; membrana tympani gone; tensor tympani normal; the entire chain drawn inwards, in consequence of the destruction of the membrana tympani.

Dr. Shaffner said disease of the mastoid cells was not an unfrequent occurrence in children, especially as a sequel to scarlet fever and measles, and that it usually terminated in recovery, even when very severe, and producing considerable destruction of bone, which is shown by the deep scars carried through life. He thought it strange that disease of structures in such close proximity to the brain does not more frequently develop inflammation of the brain during childhood, while in the adult the same condition produces such serious disease so often.

Dr. Burnett said he remembered that while attending Professor Gruber's clinic in Vienna, the fact of children being less liable to brain disease than adults, from disease of the mastoid cells, was alluded to by the professor. The reason given for this is the thinness of the outer mastoid wall in children. When inflammation occurs in the rudimentary mastoid portion of the temporal bone of a child, there is, of course, less resistance outwards towards this thin wall than inwards towards the cerebrum. Very often, when the bone is cribriform, nothing but the integument covers the abscess; and hence the great freedom of young children from fatal mastoid disease. *April 11th*, 1878.

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4. *Foreign body in the tympanum.*

Presented by Dr. H. S. Schell.

An Irishman, 55 years of age, applied at the Eye and Ear Clinic of St. Mary's Hospital for relief, on the 23d of June, 1874. He stated that while lying on the settee, at home, three days before, with his eyes closed, his wife had poured melted lead in his ear. The sudden and intense pain caused him to spring up and shake his head violently, and in this way, he said, the lead ran out of his ear again and splashed on the floor. In confirmation of his story he produced a scrap of paper containing some fragments of lead, which had evidently solidified after falling on a flat surface.

Examination of the right ear showed that the skin of the concha and external auditory meatus was deeply burned. From the depths of the meatus came a copious, flocculent discharge. When this was washed away there was found a barely perceptible rim of the membrana tympani remaining, and beyond it the space was filled with a dark-colored substance of metallic lustre. This substance extended outwards, by two projections, along the anterior and posterior walls of the meatus. A portion of the

handle of the malleus could be seen outside of the mass, showing perfectly white against the dark background on which it rested.

The facial nerve on the right side was completely paralyzed, and the patient said he had noticed the distortion of the mouth in two or three minutes after he first felt the pain. The sense of taste was lost in the right side of the tongue, and there was some dryness of the same side of the mouth. He complained of constant vertigo. He had been previously somewhat hard of hearing, but this ear, which had been his "good one," was now quite deaf. The tuning-fork on the vertex or teeth was heard in the left ear only. On the left side, in which he had been very deaf for years, the membrana tympani was entire, sunken, and opaque. The loud voice could be heard close to the auricle. There was no perceptible constitutional disturbance.

From the manner in which the lead lay in the right ear, it looked as if it would be very easy to take hold of one of the projections previously mentioned and pull the whole mass out. Although I used a very strong pair of dressing-forceps, however, and all the force I could apply, the only effect was to tear off fragments and leave the main body of the lead immovably fixed in its position. Syringing was utterly useless.

Seven days later the burned soft parts had sloughed away and left the bony canal of the meatus exposed. The foreign body was now slightly movable in its bed, but could not be extracted. After this I ceased all efforts to remove it, and merely directed that the canal should be kept clean by syringing. On the 14th of July I noticed granulations springing up around the processus brevis of the malleus. On the 21st the manubrium was hidden by the granulations which projected from above. A month later I found the inner end of the meatus crowded with granulations, behind which the lead had disappeared. I removed most of them with the looped forceps, and cauterized their bases with chloro-acetic acid, so as to give free exit to the discharge.

On the 7th of September I found the patient feeble, with a weak pulse, and complaining bitterly of the giddiness. He gradually improved during the next three months under a course of tonics. I did not see him again for two months after this, until February 23d, 1875, when he presented himself again at the clinic. The opening of the meatus was now surrounded by cicatricial tissue, and contracted to a line in diameter. A point of the lead was visible just inside of the opening. I made two free incisions through the contracted tissues, and then succeeded in extracting the lead in one piece. It weighed 18½ grains. The handle of the malleus is partially imbedded in its outer side. The inner side is a tolerably

accurate cast of the inner wall of the tympanum. A projection, about two lines in length, from the anterior side, represents the upper part of the Eustachian tube. Superiorly a small process projects towards the tegmen tympani.

Two months later I found the ear dry. A thin membrane of pearly lustre had replaced the membrana tympani. He could hear a loud voice, close to the ear, but could not distinguish words. Tuning-fork on the teeth not heard on the right side. There was no response of the facial muscles to either the induced or constant current. No improvement in taste, nor in the condition of the mouth as to dryness.

I saw the patient again a month ago. His condition remains the same. It is evident in this case that the heat of the lead must have destroyed the chorda tympani, and disorganized the facial nerve through the thin walls of the Fallopian canal.

Rau refers to a similar case, reported in the *Medicinisch-Chirurgische Zeitung*, No, 39, 1852. *April* 11*th*, 1878.

VIII.—TUMORS NOT OTHERWISE CLASSIFIABLE.

1. *Large tumor of the left superior maxilla, involving nasal and orbital cavities and frontal sinuses, removed by operation.*

Presented by Dr. J. Ewing Mears.

Dr. Mears presented the specimen of a large tumor of the left superior maxilla, removed from a patient in St. Mary's Hospital.

Dr. Clunas, resident physician, obtained the following history:

"Jas. R. D., æt. 46 years, bricklayer by occupation, of moderately temperate habits, healthy and robust-looking. Family history in general was good. He had heard that his great-grandmother had what was called a cancerous affection of the face.

"In or about the year 1864 his attention was for the first time directed to a growth in the left nostril, about the size of a pea, and apparently pedunculated.

"Six or eight months afterwards, the growth not having appreciably increased in size, an attempt at its removal was made, but abandoned on account of hemorrhage.

"Patient was able to continue at work until two and a half or three years ago, the tumor meantime increasing in size, gradually pushing its way outward and upward, forcing the eyeball from its cavity over towards the zygoma.

"During this time the patient had employed large quantities of proprietary medicines, also poulticing the growth.

"The tumor itself had been painless, although at times he had experienced neuralgic pains attributable to pressure, and also complained of pain from conjunctivitis. No ulcerative tendency had been exhibited. Occasionally slight hemorrhage had taken place from the left nostril. Formication was present.

"Within six months the entire alveolar process of the left jaw had been involved, the teeth becoming displaced. Eighteen months previous to his application for relief at the hospital, the second and third molar teeth had been extracted, being very much decayed."

The area of the growth was from half an inch to the right side of the median line of the face to a point one and a half inches in front of the

lobule of the left ear, measuring in this direction seven and a half inches. From above downwards, extending from one inch above the line of the infra-orbital border to within one inch of the lower border of the lower jaw, it measured six inches.

The external surface of the tumor was reddish, the integument being filled with small venous radicles. It was also lobulated (especially towards the inner portion), soft and elastic, this elasticity being more marked at some points than at others. At the extreme upper border externally a thin plate of bone could be felt.

After consultation with Dr. S. H. Griffith, attending physician to the hospital, who kindly referred the patient to me, I determined to remove the tumor. For this purpose the incision suggested by the late Sir William Fergusson for removal of the upper jaw was employed, and the growth fully exposed. So entirely was the osseous tissue destroyed by the tumor, that it was only required to saw through the hard palate, the mass being then readily turned out of its place. It was very soft and friable, portions of it being detached by the slight effort made to dislodge it. It was found to have penetrated the orbital cavity, completely displacing the eyeball, entered the frontal sinuses and expanded these cavities to a marked extent. Absorption of the vomer and the left lateral mass of the ethmoid bone had taken place, so that the growth came in contact with the floor of the cranium. On examination this surface was found soft and easily broken down. The hemorrhage was very slight, but one vessel requiring ligation. The patient recovered well from the shock. The external wound was closed almost entirely by adhesion, and the internal surfaces were healing rapidly, when meningitis supervened on the twelfth day and caused death. To Drs. Griffith and Clunas I am greatly indebted for the faithful manner in which they conducted the after-treatment in this case.

September 27th, 1877.

Report of the Committee on Morbid Growths:

"The specimen presented by Dr. Mears, removed from the superior maxillary region, by microscopic examination is seen to consist of epithelial cells, grouped in alveolar spaces; which spaces are formed by thin bands of fibrillar connective tissue. The cellular element is greatly in excess of the fibrous; from which, and the grouping of the cells in alveoli, the growth may be considered a carcinoma, variety encephaloid."

October 25th, 1877.

2. *Specimen of large ulcer of the neck, involving the submaxillary gland, removed by operation.*

Presented by Dr. J. Ewing Mears.

Dr. J. Ewing Mears presented a growth, in a state of ulceration, which he had removed from the neck of a patient in St. Mary's Hospital.

The patient, 30 years of age, a laborer in the coal-mines, states that three years since his lower lip was severely frost-bitten, for the relief of which caustic agents were applied. Following this condition of the lip, a small ulcer appeared at the left angle of the mouth on the lower lip.

About six months after the lip was frost-bitten he was struck by the handle of a shovel on the left side of the neck. A small swelling appeared two days after the receipt of the injury, which grew to the size of a pigeon's egg, when it broke and discharged a thin, bloody fluid.

The ulcer on the lip increased in size, and was removed in April of this year. An attempt was made by the surgeon to remove the growth in the neck at the same time, but the operation was not completed. Caustics were applied to remove the portion not excised by the knife. The wound in the lip healed promptly, and thus far there is no sign of a return of the growth. The wound in the neck did not entirely heal, a small ulcer remaining. During the summer this began to increase in size, and the patient, becoming alarmed, returned to the city and came to St. Mary's Hospital for relief.

On examination the growth was found to be as large as a hen's egg, penetrating deeply into the structures of the neck, and occupying to a greater or less extent the superior carotid and submaxillary triangles of the left side of the neck. The carotid artery in its superior portion was covered by the tumor, but did not impart a pulsatile character to it. In the act of deglutition the growth moved with the larynx, in this manner indicating its attachment to it. The ulcerated surface was about the size of a half-dollar, and discharged laudable pus. Three weeks after admission the growth was removed, the dissection required to accomplish this being very extensive, and exposing the entire anterior triangle of the neck. It was necessary in this dissection to remove the sheath of the carotid artery and internal jugular vein, with the descendens noni nerve, the anterior portion of the omo-hyoid muscle, the submaxillary gland, and finally to detach it from the larynx. The facial and lingual arteries, with some smaller branches, required ligation. The hemorrhage was not great, and was easily controlled.

The large wound left was very much reduced by sutures, and the cavity was packed with lint saturated with carbolized oil. At this time the wound is rapidly filling up with healthy granulations. *September 27th*, 1877.

Report of the Committee on Morbid Growths:

"A microscopical examination of the specimen presented by Dr. Mears, shows at and around the ulcerous part an inflammatory infiltration into the connective tissue. A section taken from the deeper portion is seen to consist in part of lymphoid cells, characteristic of a sarcoma.

"An examination of the submaxillary gland shows it to be the seat of an inflammatory infiltration." *October 25th*, 1877.

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3. *Tumor involving submaxillary gland, removed by operation.*

Presented by DR. NANCREDE; notes by DR. FENTON.

J. McB., æt. 47 years. Occupation, dyer. General condition, very good. Admitted to the Protestant Episcopal Hospital September 17th, complaining of a dense hard growth (three inches by two and a half inches) situated in left submaxillary space and adherent to the bone. First noticed three months since as a small "kernel" in the neck. No pain. Two years since, an epitheliomatous growth (one inch square) of two years' duration was removed from the centre of the lower lip. This tumor caused pain by its presence. One year since, another of these tumors, about half the size of a walnut, was removed from the lower lip a little to the right of the left commissure. He states that his maternal uncle had such a growth on the lower lip. It was extirpated and never returned. There is no history of syphilis. The specimen was removed September 18th, 1877, along with two inches of jaw-bone. The patient has since done exceptionally well. *September 27th*, 1877.

Report of the Committee on Morbid Growths:

"A microscopical examination of the tumor presented by Dr. Nancrede, and referred to Committee on Morbid Growths, is seen to consist of fibrillar connective tissue, grouped into bundles of various sizes. The cells are comparatively few in number, spindle-shaped and nucleated. The growth may be classed among the connective-tissue fibromata. An examination of the gland removed with the tumor shows an infiltration of a similar character." *October 25th*, 1877.

4. *Cystic tumor in the axilla.*

Presented by Dr. W. W. Keen.

James McAndrew, æt. 27, Irishman, from Shamokin, miner, admitted to St. Mary's Hospital October 20th, 1877. He states that three years ago he first noticed a small lump in the right axilla, which at first grew slowly, but of late quite rapidly. No pain was ever experienced, and the mechanical inconvenience is the only reason for seeking aid. The tumor measured about ten inches across. It extended from high up in the axilla to the ninth rib, and ran under both the pectoralis major and latissimus dorsi. The veins were somewhat enlarged. It was apparently divided into two parts by a marked indentation, but palpation showed that fluctuation not only existed on each side of the apparent division, but from one side to the other. The diagnosis of unilocular cystic tumor was made, and confirmed by obtaining a small part of the fluid with a hypodermic syringe. The fluid was opaline, and by heat and nitric acid the amount of albumen was large. Considerable bleeding followed at the point of puncture, though no visible vein was injured.*

October 24th, 1877.—A straight and, in the lying posture, nearly vertical incision, five inches long, was made. After the skin and fascia were divided and the surface of the tumor reached, the knife was laid aside, and the cyst was gradually peeled loose entirely by the fingers, and removed intact, though the walls were very thin. Not a single blood-vessel was ligated. The axillary artery was readily felt at the apex of the wound. Four sutures, adhesive straps, and two compresses completed the dressing. Except a slight tendency to burrowing downwards, readily met by drainage, the man did excellently, and went home with the wound nearly healed, November 7th.

The cyst proved to be unilocular, with reddish, thin liquid contents. The sac consisted of a thin, transparent wall, thickened at numerous points by thin plates of almost cartilaginous hardness. The apparent septum was due to a tight band of connective tissue, which constricted it externally.

October 25th, 1877.

Report of the Committee on Morbid Growths:

"The fluid contained in the cystic tumor presented by Dr Keen, upon

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* To this bleeding was probably due the different color of the fluid found after the operation.

microscopic examination, is seen to contain compound granular cells, leucocytes, squamous epithelial cells, shreds of fibrin, numerous cholesterin crystals, and amorphous granular matter. By the employment of the nitric-acid test, albumen was found to be present in large amount."

November 8th, 1877.

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5. *Tumor removed from the parotid region.*

Presented by Dr. John H. Packard. Notes by Dr. Robert M. Smith.

Robert J. Ray, æt. 58; Irish; by occupation a ship-carpenter, was admitted to the male surgical ward of the Episcopal Hospital, October 26th, 1877. Stated that nearly thirteen years ago he noticed a small, firm, painless nodule forming in his left cheek, just above the angle of the jaw. It was freely movable, and gradually increased in size, until it became as large as a hen's egg, but never interfered at all with the motions of his jaws, or gave him any pain, until about two weeks ago, when he suffered from a severe shooting pain in the tumor and left side of his face. He then noticed that his face was drawn to the right side, and, as he says, was without sensation; the left eye also, at this time, "became crooked."

When admitted to the hospital, there was a firm tumor, slightly but distinctly movable, occupying the whole of the left parotid region, and extending under the ramus of the jaw, not sensitive to pressure. Paralysis of the muscles of expression of left side, visible even when the face is at rest; no loss of sensation detected; paralysis of the superior oblique muscle of left eye; tongue protruded straight; no interference with movements of jaw.

Tumor removed October 29th, 1877. It was found to lie directly upon the sheath of the carotid artery, implicating, to some extent, the parotid gland, the duct of which had to be divided. No hemorrhage occurred during the course of the operation. Wound closed, and dressed with lint soaked in laudanum; morph. sulph. gr. ½, hypodermically. Liquid diet.

November 3d.—Wound looks remarkably well; no pain; ligature came away; stitches removed; placed on soft diet. Says he feels very well, and wants to go home.

November 4th.—Had a slight chill.

November 6th.—Cough; pain in the chest; physical examination revealed pneumonic consolidation of anterior portion of lower lobe of right lung. Sputa tinged with blood, and very tenacious.

November 7th.—Very little febrile reaction; no extension of pneumonia.

November 22d.—Wound of operation almost wholly healed. The pneumonic symptoms nearly gone. *November 22d*, 1877.

Report of the Committee on Morbid Growths:

"The tumor removed from the parotid region presented by Dr. Packard, and referred for examination to the Committee on Morbid Growths, is found to be composed of different histological elements, constituting several varieties of new formations. In circumscribed localities are seen epithelial cells, grouped together irregularly within alveoli, formed of fibrillar connective tissue, constituting carcinoma,—variety scirrhus. In other parts of the growth are seen large round cells, which contain a large granular eccentric nucleus. The cells appear to be imbedded in a hyaline matrix, through which run a few isolated fibres of fibrillar connective tissue, characteristic of cartilaginous tissue. The remaining and greater bulk of the tumor is seen to consist of spindle, stellate, and oval cells, with a fibrillar connective-tissue intercellular substance. Here and there the cells form a reticulated tissue, in the meshes of which is seen a structureless substance, made granular by the employment of the reagents used in preparing the section; a myxomatous tissue. The blood-vessels throughout the growth are very distinct, and have their walls somewhat increased in size by concentric layers of fibrous tissue. The new formation may be considered a carcinoma myxomatodes, containing cartilaginous tissue."

December 13th, 1877.

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6. *Recurrent epithelioma of neck.*

Presented by Dr. C. B. Nancrede.

Dr. Nancrede said that he expected to show a specimen of such rarity as had never been presented during the more than twenty years' existence of this Society. As far as he knew, there were but three other such upon record, related by Sir James Paget in his classical work on Surgical Pathology, the accounts of which had been read at the previous meeting but one. Only two of these had been subjected to such thorough scrutiny as to leave no room for doubt. Unfortunately, like so many other "unique cases" and "original discoveries," this one, upon more rigid examination,

had caused the "châteaux en Espagne" to vanish into thin air, and had left only an exceedingly commonplace specimen. Dr. Nancrede said that he trusted, however, this mistake would prove, as is so often the case, more instructive than any number of rare specimens. It may serve as an admirable commentary upon the remarks made in relation to the case just presented by Dr. Cleemann, and the one adverted to by Dr. Tyson. The examination of these specimens by competent gentlemen apparently resulted in most diverse opinions as to their nature. In Dr. Cleemann's case, the Committee on Morbid Growths examined only the skin, and found epithelioma, a fact that it gave Dr. Nancrede great pleasure to corroborate by his own examination of their sections. But upon examination of the deeper tissues, Dr. N. found true carcinoma. Dr. Tyson examines the portion of the growth sent to him, and finds nothing but adipose tissue, while the real morbid growth is sent to another gentleman, who of course discovers its true nature, because he has the real growth, while Dr. Tyson had nothing but the subcutaneous tissue to examine. In the last case, Dr. Nancrede again confirmed the accuracy of the committee's report upon the *section they examined*, in which he could discover nothing but of a fibro-cellular nature. When Dr. Simes told him that the specimen before the Society this evening was a most beautiful specimen of epithelioma, he was driven to one of two conclusions, viz., that the committee, whose section he had not then seen, had made a mistake, or that he was on the eve of a most important pathological discovery, which was that *epithelioma* and so-called *fibrous cancer* were interchangeable. He naturally proceeded to re-examine the first growth. Taking the portion left imbedded by your committee, and, after levelling the surface, removing a thickness of perhaps a sixteenth of an inch, he made three sections, all of which, to his astonishment, were typical specimens of epithelial cancer. Here two strokes of the knife would have prevented any mistake in the original examination.

Dr. Nancrede then read the following notes of the case:

For the history of the first growth I must refer to the notes presented with it on the evening of September 27th, 1877. The specimen before the Society was removed, January 2d, 1878, from John McB., æt. 48 years, by trade a dyer. It consists of the contents of nearly the whole anterior superior carotid triangle, the large wound resulting from which, coaptated by harelip pins and sutures, has united well, except at its central portion, where skin was wanting for its complete closure. McB. states that the wound healed in about three weeks,—my term of hospital service having elapsed before his complete convalescence,—leaving, however, a

suspicious tubercle at the spot originally left open for drainage. This nodule became painful and commenced to grow rapidly some three weeks after complete healing had occurred. After three weeks of rapid increase it apparently ceased to grow, at least superficially, remaining in the same state from the beginning of December until the 27th of the same month, when I first saw him. The growth was then about the size of an English walnut, with the surrounding tissues somewhat infiltrated, and firmly adherent to the old section of the lower jaw. It also was so firmly attached to the hyoid bone and thyro-hyoid membrane as to give considerable trouble in its removal, in which I was kindly assisted by my friends and colleagues Drs. Henry, Simes, and H. Reed. *January 24th*, 1878.

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7. *Recurrent carcinoma.*

Presented by Dr. Richard A. Cleemann.

The morbid growth which I exhibit to-night is the third of a series removed at different times from the axilla of the same individual, a lady about 70 years of age, the primary and secondary ones having already been presented before the Society a year ago (January 11th, 1877).

The initial one of these growths was first observed, as I stated on the former occasion, as a small "lump" in the axilla, some time during the spring of 1871. Having increased in size, and presenting the gross appearances of *epithelioma*, it was removed by excision, four years subsequently, April 2d, 1875. The diagnosis of epithelioma was confirmed by your Committee on Morbid Growths, by which it had been submitted to microscopic survey: my friend, Dr. C. B. Nancrede, who made an examination of the base of the tumor, found the elements of *scirrhus*. A tedious convalescence, interrupted by sloughing of the cellular tissue adjacent to a part of the wound, followed; but eventually the patient was restored to a condition of excellent health.

A year after the excision a hard nodule began to show itself near the seat of the primary growth. This second morbid mass was taken away January 3d, 1877, and, being submitted, like the first, to your Committee on Morbid Growths, was declared by it to present the characters of "a carcinomatous infiltration of a lymphatic gland, variety *scirrhus*." The convalescence from this operation was somewhat less prolonged than that following the first, and was complicated by an attack of superficial erysipelas, not invading the skin about the wound, but extending over other

portions of the surface during a period of more than two weeks. In connection with this process, small collections of pus, healthy in appearance, formed under the edges of the wound, and a swelling of the cellular tissue in the most dependent part of the arm was observed, the latter remaining as an œdematous infiltration of the tissue after the erysipelas had run its course. After this complication, I noted in my case-book that the patient's skin had a waxy aspect, similar to what was formerly described as belonging to the "cancerous cachexia;" but this appearance no longer existed when good health was again established.

In the latter part of August, 1877, the patient, at that time in most excellent general health, became conscious of a new growth situated in the neighborhood from which the others had been removed. She consulted me two months subsequently, when the mass could be felt to be about the size of a small marble: it was situated high up in the axilla, immediately anterior to the scars left by the incisions made for the removal of the former growths. It was hard, of irregular contour, and closely attached to the skin above it. At the same time the cicatricial tissue itself was found to be adherent to the subjacent parts. The patient suffered occasionally from pain in the left arm, and had lately had a tingling sensation extending down the extremity to the little and ring fingers. The swelling before referred to as existing in the arm had never entirely disappeared. This, the third growth, lies before you, having been dissected away on November 17th, 1877, since which time it has been preserved in alcohol. It was found to lie directly upon the subscapular artery, not far from its origin in the axillary. So close was it to the subscapular that Dr. Agnew, who performed the ablation, judged it best to throw a ligature about that vessel, fearing that its coats had been damaged by the knife. The mass now resembles a small cluster of somewhat enlarged lymphatic glands attached to a portion of skin by indurated cellular tissue. Dr. Nancrede, who was good enough to examine it microscopically for me, has found the glandular part to possess the *carcinomatous* features of the immediately preceding growth (*scirrhus*), the skin to be normal, and evidences of inflammatory action in the connective tissue. The wound from this last operation has been healing kindly, with little suppuration, though the loss of tissue in the previous excisions has contributed to a slow contraction of its edges. It is now almost closed, but a short linear ulcer remaining. Save some constitutional disturbance immediately after the operation, the general health on this occasion has not been affected.

But the removal of the growth has not been followed by a disappearance of the occasional pain and tingling in the affected extremity, nor has the

œdematous swelling vanished, while the development of a small, crack-like ulcer in a depression of the old cicatrix, the tissue about it being red, shiny, and "cushiony," is additional evidence of the remaining activity of the disease.

The clinical facts with which I have supplemented the brief description of the morbid anatomy of the above case, have been introduced because they illustrate so forcibly the pathological question of the local nature of carcinomatous disease. Here is a delicate and aged lady who has suffered for seven long years from cancer, and yet is as strong now in her general health as when the disease first showed itself, seeming indeed to have escaped the full measure of senescence which is wont to accompany that lapse of time. Moreover, the complications in the convalescence from the first two operative procedures, while they brought her for a season to a condition of great prostration, went for nothing in urging on the march of the disease, if they did not positively, in their destruction of neighboring compromised tissue, rather stay its ravages.

The combined presence of epithelioma and scirrhus, with the recurrence of the latter alone, is worthy of notice. Following Virchow and those who believe that the cancer-cells may originate from other than epithelial cells, the pathologist may look upon one suspected irritant as producing here the two forms of carcinoma. Exerting its power on the epithelial layer of the skin, epithelioma was developed, but when it bore upon the connective tissue, scirrhus was the result. The more circumscribed nature of epithelioma made the entire ablation of its tainted cells practicable, and this form of disease did not recur. On the other hand, the infiltration of the elements of scirrhus, spreading more widely, had left some seed behind to bring forth its proper growth.

Dr. Longstreth said, in every case when the metastatic formation of a tumor takes place, either after an operation for the removal of a new growth, or when no surgical interference has been made, the questions must rise, first,—Is secondary growth the same, and does it always show the same histological characters, as the primary? and, secondly,—What is the dependence, or connection, or relation, of one on the other?

In regard to the first question, it must be answered, certainly in the great majority of cases, in the affirmative. A careful study of microscopic sections from the respective growths, shows such correspondence of the cells and their relations, that without doubt the secondary growth is placed in the same class, and given the same name, as its predecessor. In some cases, however, the similarity is not so close, and there does occur a diversity in respect to the structure of two new growths, which growths give the same

reasons for belief that they bear the relation of parent and offspring, that we have in cases where similarity in structure exists. Under these circumstances, a subordinate question arises, whether these new formations do have such relationship, or whether we are dealing with an instance of two distinct varieties of malignancy occurring in the same patient, the two growths not, however, interdependent, but each growth separately developed in a distinct position and at different times. The possibility, nay, the certainty, of such distinct development must be admitted, although common experience turns our attention almost wholly to the question of metastasis.

It is probable, without, perhaps, entire definiteness of statement being possible in the matter, that a consideration of the different grounds or mother-beds, in which the two growths respectively have their origin, may help us to explain the variation in appearance presented by tumors metastatically related to each other, and undoubtedly belonging to the same class. Such variations in appearance may be illustrated—although the illustration does not prove the point in question—by the microscopic results derived from the examination of newly-developed granulation-tissue, and of the same tissue in a state of luxuriant growth; or from what is seen in syphilitic growth before and after ulceration of the covering skin; or, to take an instance more nearly allied to the question in hand, the appearance of a newly-developed and growing sarcoma, and the condition presented by tumors of this class at a late stage of their growth. As a further illustration of the differences existing between the primary and the secondary metastatic growths, he would cite a case studied by Prof. Brodowski, and reported in *Virchow's Archiv*, Bd. lxvii., in which was found melanotic sarcoma of the eye, with apparently carcinomatous tumors of the liver and kidney.

Besides the differences due to the dissimilarity of tissue, in which the primary and secondary growths have their seat, for example, a tumor of the brain secondary to one of the mammary glands, the variations in appearance may, and undoubtedly do, result from mere difference in age, and rapidity of growth. He did not think that scientific oncology is capable at the present time of furnishing us with sufficient data for the determination of these essential points.

The second question, viz., the dependence or connection of the metastatic offspring from the primary growth, leads to the consideration of the means by which this is effected. The two possible channels of communication are the blood- and lymph-circulations. Although these two means have long been pointed out, yet it is only within quite recent times that com-

pletely developed tumor-cells have been shown actually in transport by these roads, and actually free within the calibres of both these vessels. Let us leave to one side, for the present, the consideration of that as yet indefinite idea of the conveyance of germs by these means, which germs may or may not develop as a distinct growth. The separation of these two ideas is the separation of the question of local disease, and of cachexia in malignant tumors. Accepting now the first of these two theories as the one definitely proved, viz., that, starting with the local growth of a tumor, we may have metastasis, or the development of secondary growths in other parts, through the bodily transport, in a developed form, of parts of such growth, the question arises,—In what forms and at what parts does such secondary development occur?

The now common knowledge, backed by many careful observations, of these travelling masses of tumor, in both the lymph- and the blood-track, leads us to study the development of metastatic growths in both of these situations. The possibility of the passage of cells through the walls of the vessels within the tumor, vascular walls that are no longer normal, seems to be acknowledged; the passage of the cell-elements into the lymph-vessels, along with the fluids or juices of the tumor, is well established. The masses, which can be called tumor-emboli, are carried along the blood-current until they find a suitable resting-place. This seat may be determined by their attachment or adhesion to the wall of a venous trunk, or by what is more likely to happen, their passage through a capillary net-work, which by its size filters out the little masses. The position, therefore, of their future growth depends somewhat on the relative disproportion in the size of the embolus and of the capillary vessel. Similar emboli are found in the lymph-capillaries.

The most frequent of metastases, and the one with which we are the most familiar, is the implanting of the tumor-growth on a neighboring lymphatic gland. Here the infection of the gland likewise takes place through the direct transport of the cell-elements of the new growth. Here, too, sometimes, without doubt, we see changes effected in the typical histological appearances of the tumor-cells as a result of their implantation on glandular tissue.

In addition to these two modes of metastasis, in both of which the material transported for the growth of secondary formation is the developed cell, separated as such from the primary growth, there is at least another mode, which is a possible one, if not probable, by which the dissemination of diseased tissue-elements can be effected. Let us look at the circumstances under which the primary growth has its beginning. As the initial

step there occurs at the seat of the disease a mal-growth of the nuclear bodies from which are derived the heteroplastic cells of the new formation, and which become the nuclei of the tumor-cells. The nuclear bodies are either the newly-deposited elements or the derivatives of cell-division of the tissue previously existing. In either case, according to theory, they originally existed as normal (white) corpuscular elements of the blood, and which, under local morbid influence, have had their developmental capacity so far altered that, instead of presenting the "fixed appearance" of health, we see, for example, an epithelial element becoming a carcinoma, or a connective-tissue element a sarcoma. Now, these normal blood-corpuscular elements possess the formative power of growing, in the one case, as supposed, epithelium, in the other case, connective tissue, but under local morbid influence develop into heteroplastic growths. We see, now, either one of two facts, either that only such corpuscular blood-elements find their way into a particular tissue, or that only such of these wandering bodies develop in the part with which they are in correspondence; all other wandering elements which may chance to find their way into, let us say, connective tissue, but which have not the power of developing into that tissue, are carried back by the lymph-vessels to the general circulation.

If we have, then, normal corpuscular elements which only find development in tissues to which they are histogenetically allied, the same rule of development must hold for the abnormal corpuscular elements, viz., the nuclei which are capable of growing tumor-cells. The abnormal elements, the undeveloped cells, the nuclei, which exist in abundance in the morbid growth, are free to move, and are liable to be, and are constantly, taken up by lymphatic agency, to be reconveyed into the general circulation. Such nuclear bodies can exist in the blood, and, from their similarity to the white blood-corpuscle, remain undetected. They must now be looked on as derivatives of a particular tissue, and as bodies having impressed on them a morbid formative power; they too, like their prototypes the normal elements, seek their place of development in the tissue to which they are allied histogenetically.

This process, which Dr. L. had attempted to characterize, is, he wished distinctly to state again, something very different from that which has been alluded to in embolic metastasis of tumor-cells, and from the infection—the word is used without prejudice of meaning—of a neighboring lymphatic gland. The value of such an hypothesis for metastasis is to be found in cases where secondary formations occur, either in parts void of tissue histogenetically related to the new formation, or when the embolic method

can be excluded from participation in the process. As an instance of the first may be cited an enchondroma of the lung, secondary to such a tumor of the testicle. An instance of the second would be with difficulty found, and with greater difficulty proved. This hypothesis is something very different from embolism: the one is growth within the tissues of an organ, and in a particular tissue, the latter is growth at a fortuitous point with a blood-vessel.

According to the terms of the hypothesis, the metastatic formation of an epithelioid growth would take place in an epithelial structure: thus, an epithelial growth of the stomach, when it involves the liver in secondary formations of the same malignant character, chooses the liver-cells—cells which, if they are not truly epithelial in character, bear at least a close relationship to such tissue—as their point of development, rather than the connective tissue of the portal canals, etc. (excluding, of course, cases of metastasis between the two organs by embolism); whereas in sarcomata the point of development would be the connective-tissue parts of the organ. In Brodowski's case, already cited, the author speaks of the knots in the liver, metastatically related to the melanotic sarcoma of the eye, presenting the structure of so-called medullary carcinoma. (See *loc. cit.*, Pl. VI., Fig. 2.) In Fig. 3, Pl. VI., the drawing represents a connective-tissue tract bordered by areas of liver-cells; the connective tissue shows in its midst, or is replaced by, the melanotic sarcomatous formation, whilst the liver-cells, collected in clumps or masses surrounded by a very delicate stroma, exhibit the changes which the author considers as carcinomatous. The liver-cells undoubtedly show alterations, but he doubted if they represent or are to be considered as the part typical of the new formation in the organ; he doubted, too, if the delicate connective tissue enclosing them is to be regarded as a stroma of a glandular carcinoma. Much more likely is it that the stroma is but the extension inwards between the acini of the connective tissue of the portal canals, either as a hyperplasia or as a malignant new formation, connected with the melanotic sarcoma distinctly developed in those canals. The condition of the liver-cells differs but little from, and is parallel to, that seen in cirrhosis of the organ. The relative changes of the two tissues—on the one hand the liver-cells first showing alterations from proximity to the irritative process present in the adjacent tissue, but finally being destroyed by the overgrowth and contraction of such tissue, and on the other hand the connective tissue becoming hyperplastic and tending constantly to invade the areas of the liver acini—are the same in that disease as we see here represented in the malignant growth. This explanation of the appearances of the liver in Brodowski's case—and

the same is true of the kidney changes—affords a confirmation of this hypothesis.

Dr. L. mentioned that recurrence of tumors, in contra-distinction to metastasis, has the same significance as the extension of the growth in its original formation and enlargement, and takes place by peripheral infiltration; although, in the removal of a tumor by surgical operation, the incision may be carried through apparently normal tissue, yet if recurrence takes place there is proof either that some of the surrounding part was involved, though invisibly, or that the morbid formative character was so impressed on the cells, that they had become incapable of development in the normal type. *January 24th*, 1878.

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8. *Tumor over the superior maxilla.*

Presented by Dr. Harrison Allen.

Miss A., æt. 24, noticed, in September, 1877, a pimple over the superior canine tooth. Soon afterwards she noticed swelling of the face of the same side. When first seen, through Dr. Sinkler, the swelling was rounded, sessile, one-quarter of an inch long, situated at the junction of the lip and gum, on the right side of the frænum, and extending thence across the frænum. A small fistule was seen on a teat-like eminence, which, however, did not receive anything larger than an Anel's probe. The parts were freely excised, hoping to come upon the sac of an alveolar abscess. No such structure became apparent, the parts cutting like cartilage. Inasmuch as the parts continued to increase in size, the growth was removed under ether, February 17th, 1878, Drs. Sinkler, Keen, and Cryer assisting. The swelling in part had returned by March 16th, the patient complaining of a pain as though the parts were occasionally stung by a bee.

March 28th, 1878.

Report of the Committee on Morbid Growths:

"A microscopical examination of the specimen presented by Dr. Allen shows it to consist of a dense fibrillar connective tissue, infiltrated with numerous small round lymphoid cells. Most probably the growth is the result of an inflammatory process." *April 11th*, 1878.

9. *Cylindrical epithelial carcinoma of the omentum.*

Presented by Dr. S. W. Gross.

A tailor, 50 years of age, was admitted into the Jefferson Medical College Hospital, November 7th, 1877, on account of a tender, hard, globular, subcutaneous tumor, which was seated in the linea alba, and evidently communicated by a pedicle with the interior of the belly. The man was thin, weak, and sallow, and broken down by previous suffering.

He stated that he had been affected for six weeks with bronchitis, and burning pains in the umbilical region, the latter of which, having become continuous and fixed for three days, led him to detect the circumscribed swelling on the 7th of August. In the interval he had lost flesh, strength, and appetite, and the suffering was aggravated by the ingestion of food. The bowels were inclined to be costive; there was no vomiting; and the burning pain, which, along with emaciation, and loss of appetite and strength, was the prominent symptom, lasted up to the date of operation, being quite violent at times, but particularly after eating, and frequently depriving him of sleep.

On removal, the tumor was found to lie on the aponeurosis of the external oblique muscle, and to be connected, through an opening in the linea alba, with the omentum by a rounded pedicle one-third of an inch in diameter and half an inch long. A ligature having been cast around the foot-stalk, the mushroom-like mass was cut away, and the wound dressed in the ordinary manner. After the operation the appetite improved, and the pain was greatly diminished, and he was discharged at the expiration of a fortnight. Ascites, however, gradually made its appearance; the improvement was only temporary, and death ensued on the 14th of January, 1878. Unfortunately, a *post-mortem* inspection of the body was denied.

The neoplasm was one inch in diameter, moderately firm to the touch, finely nodulated, and surrounded by a delicate capsule of loose connective tissue and fat, which dipped in between the nodules. On section, the surfaces were white and alveolated, the spaces being separated by bands of fibrillar connective tissue, wide in proportion to the diameter of the alveoli, which were always minute.

On microscopic examination, for which and for the drawing I am greatly indebted to Dr. E. O. Shakespeare, thin stained sections presented pictures which corresponded to the descriptions and illustrations of many authors of cylindrical-celled epithelioma. "The great mass of the tissue consisted

of wide bands of parallel bundles of connective tissue in a state of inflammatory irritation, and passing in every direction. In these, and

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parallel with their course, were the blood-vessels. In many portions of this mass were seen rather numerous roundish alveoli, the walls of many of which were lined with a single layer of well-defined columnar epithelium, consisting usually of a protoplasm slightly granular, a single, large, double-contoured nucleus, located nearer the attached than the free extremity of the cell, and often also of a thickened line forming the free or butt end of the cell. The diameter of the latter was usually about one-fourth to one-third of its length. The walls of the alveoli were only partially covered with this single layer of epithelium, and the cells, instead of always exhibiting the characters above described, were sometimes greatly swollen, and almost entirely filled by a colloid substance. The lumen or central portion of the alveolus was either entirely empty, or filled, partially or completely, with an amorphous, transparent, colorless material, containing a few scattered minute dark granules and cellular detritus. The walls of a few of the alveoli were entirely denuded of their covering of cylindrical cells, the lumen being filled with a colloid mass, interspersed here and there with epithelium, in a state of colloid metamorphosis. In addition to the round and oval shaped alveoli, tubes, which were also lined with a single layer of columnar epithelial cells, were often seen. Many of these channels, both the alveolate and tubular, were in communication with one another."

From the stand-point of pathological histology, it is interesting, in the absence of a *post-mortem* examination, to determine whether the neoplasm was merely a secondary deposit in the omentum, or whether it was a primary heteroplasia of the elements constituting that organ.

In an elaborate paper, "Ueber Carcinoma Peritonæi,"* Dr. Theodor Petrina has collected forty cases, of which fourteen were primary and twenty-six were secondary growths of that membrane. Of the former, nine were encephaloid and five were scirrhous tumors, of the latter, fourteen were encephaloid, ten scirrhous, and two colloid formations; the primary disease having been seated in the stomach and pyloric end of the duodenum in seventeen, in the ovaries and its tubes in four, in the liver in three, and in the pancreas in two cases.

To those who believe, with Waldeyer, that carcinoma always has an epithelial origin, the above statistics are worthless; since in primary carcinoma of the peritoneum the only elements that can participate in the proliferation are the endothelial cells covering the connective-tissue network, the connective-tissue corpuscles, and the nuclei of the vessels. Such accurate observers as Cornil and Ranvier,† however, describe primary carcinoma of the peritoneum, and state that it usually commences in the omentum as encephaloid, scirrhous, or, more frequently, as colloid; and Rindfleisch‡ holds the same views. These authorities make no allusion to primary cylindrical epithelioma; so that if it be assumed that the growth under consideration was protopathic, the presence in it of columnar epithelium can only be ascribed to a heteroplastic process. In support of such a view, many analogies might be adduced. Thus, H. v. Wyss§ has described a colloid cyst, situated between the abdominal muscles and peritoneum of an adult, which contained, in addition to the fluid contents, ciliated cylindrical epithelium; and similar appearances were met with in a large cyst on the posterior wall of the œsophagus, an inch and a half above the cardiac orifice of the stomach. Hence the cysts were heterologous as regards their origin, as they were found in situations in which there is no normal ciliated epithelium. In the same way the tumor which I have described may be regarded as a primary heterologous or heteroplastic production.

With the followers of Waldeyer, if the view be taken that the tumor originated in the omentum, we may ascribe its development to a germ from the glandular layer of the embryo, which was displaced and included in

* Prajer Vierteljahrschrift, Bd. 2, p. 41, 1872.

† Manuel d'Histologie Pathologique, troisième partie, p. 965.

‡ Text-Book of Pathological Histology, p. 273.

§ Virchow's Archiv, Bd. 41, p. 143.

the tissue of the omentum. In this way Dr. Kolackek* has quite recently explained the origin of a voluminous tumor of the lower jaw of a woman, aged 28 years. After removal by excision of one-half of the bone, the mass proved to be an adenoma undergoing cystic degeneration. "The structure was exquisitely acinose, the acini being lined by a layer of cylindrical cells." The occurrence of a cylinder-celled epithelioma in the omentum surely cannot be held to be more remarkable than the formation of an adenoma within the osseous tissue.

Instead of being a carcinomatous outgrowth of the omentum, the neoplasm may have been a primary cylindrical epithelioma of the transverse colon or small intestine, which, from its original seat in the mucous membrane, progressively invaded the other coats of the gut, and finally formed a polypoid nodule, which protruded through the opening in the linea alba, where it gradually increased in size. The absence of nausea and vomiting and hemorrhage, the presence of which would rather have denoted carcinoma of the stomach, and the constipation and intense suffering after the ingestion of food or during the peristaltic action of the intestines, would point to the origin of the tumor in the small intestine; although it should be stated that in the cases of carcinoma of the peritoneum, analyzed by Dr. Petrina, dyspeptic symptoms, pain, constipation, and subsequent ascites were the prominent phenomena.

Although the general history of cylindrical carcinoma of the stomach, intestines, gall-bladder, and biliary ducts is that it gives rise to secondary nodules in the liver, without involvement of its serous covering or of the peritoneum elsewhere, and although I have been unable to discover an example of secondary infection of the omentum, yet I am disposed to regard the tumor in question as a secondary formation, either by the usual way of generalization or by contact with an underlying mass, through which the endothelial cells of the omentum were converted into cylindrical epithelial elements. Whatever view may be taken of its origin, the case is none the less remarkable from a surgical stand-point, in being, so far as I can discover, the only example of removal, during life, of a cylindrical epithelioma which was developed in the cavity of the abdomen.

March 28th, 1878.

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* Centralblatt für Chirurgie, No. 38, 1877, and New York Medical Record, March 23, 1878, p. 229.

10. *Colloid cancer of the abdomen.*

Presented by Dr. Thomas M. Drysdale.

I visited Mrs. Margaret M. Rice, in consultation with Dr. William B. Atkinson, October 30th, 1877, to examine a tumor of the abdomen, which was supposed to be ovarian. She gave me the following history of her case. She was thirty-three years old; had always been healthy, except an attack which her medical attendant called "bilious intermittent fever, with liver complaint." This was twelve and a half years since.

She first menstruated when between sixteen and seventeen years old, and had no pain or trouble of any kind; has always been regular, except when pregnant, until two months ago, when she missed it, and has not menstruated since. Her menses were never accompanied with pain, were normal in amount, and usually lasted two and a half or three days.

She was married when twenty-one years of age, has had three children, all now living, the youngest three years old last May, the next six next month, and the oldest nine the 1st of October. Her labors were perfectly natural and easy. The first was slow, the others quick. She nursed all her children. After marriage she was always troubled with leucorrhœa, often quite profuse.

She has been losing flesh for over a year, and has become greatly emaciated and anæmic. She has had no trouble with her bladder; her bowels are natural in every way; her appetite remains good, and up to this time she has done her own work.

The enlargement was first noticed two years ago, in November, when nursing her child, and just at that time she had an attack of nervous prostration, which her doctor told her was owing to prolonged lactation, and advised her to wean her babe.

The swelling commenced in the centre of the lower part of the abdomen. It was comparatively small till last June, and then rapidly increased, causing her to look "just like a woman in the family way." This rapid growth has continued to the present time.

She has never had pain in the abdomen until two months ago, and then for a short time had severe shooting pains, which were supposed to be owing to an umbilical hernia. She was first attended by Dr. Ruttey, who used electricity. She then went to the University Hospital, where she was examined by Dr. Goodell and told she had an ovarian tumor. Dr. William B. Atkinson was next called upon, and he, in company with Dr.

I. Williamson, tapped her, and obtained about a quart of a jelly-like matter. These gentlemen also diagnosed an ovarian tumor,

I found the abdomen very prominent and enormously distended. A large umbilical hernia existed, filled with fluid, a contraction in the middle causing it to appear double. The abdomen was dull on percussion, except along the region of the ascending colon. The right flank was resonant, the left dull, and along the edge of the ilium semi-resonant. Fluctuation could be detected everywhere, except in the right hypochondriac region. The tumor was smooth, elastic, and immovable. There was neither pain nor tenderness on pressure.

Examined per vaginam, the uterus could be felt low down, with the cervix pushed towards the right side, and pointing that way. The uterus was immovable. The sound entered three inches, and so anteriorly that the handle had to be pressed against the perineum to insert it. The back of the pelvis was filled with hard, fixed masses, which felt like uterine fibroids, and were tender to the touch. The uterus was intimately connected with or imbedded in these growths.

December 8th, 1877.—In company with Dr. W. L. Atlee and Dr. Atkinson, I tapped her, and removed about three quarts of a colloid matter, which resembled in color, consistence, and general appearance calf's-foot jelly. There also escaped small quantities of a thin greenish fluid. It required fully an hour to obtain this quantity, although a large canula and trocar were used.

After tapping, the epigastric region and whole upper part of the abdomen became resonant on percussion. On deep pressure, a hard mass could be felt in the left hypochondrium. Palpation gave the sensation as if a stratum of thin fluid existed between the abdominal wall and tumor. The mass could now be moved to a certain extent. My diagnosis was given as colloid cancer of the abdomen, and an operation declined.

The tapping relieved her of the feeling of distention, and on visiting her the next day I found her sitting up. Her color had improved, her lips were red, and she said she felt quite comfortable. Dr. Atkinson now took entire charge of her case. She continued to emaciate, the abdomen enlarged, and in a month or two the umbilicus ulcerated, an opening forming, through which large quantities of colloid substance, and a greenish fluid, were discharged, as much as a bucketful of the last escaping at one time. When I last saw her, June 5th, the abdomen had entirely changed in appearance; the lower part had retreated, more especially on the right side, leaving a hard mass filling the entire upper part of the belly, and another and softer mass the left lower portion. She died June 8th, 1878.

Assisted by Dr. W. L. Atlee, Jr., I made an examination of the body June 9th. The emaciation was extreme. The abdominal wall was so thin that my first incision penetrated the cavity. The peritoneum was thickened and opaque; its surface was unevenly studded with white deposits, resembling the diphtheritic membrane; the subperitoneal areolar tissue was converted into colloid, forming a layer from an eighth to a quarter of an inch in thickness. There was no organ which had a natural appearance. Filling the epigastric, the right and left hypochondriac, and left lumbar regions was an enormous colloid tumor, which careful examination proved to be the degenerated omentum. When cut, it had the appearance of a honeycomb, the cells or alveoli being filled with a pale-yellow jelly. Below this mass were the small intestines, lying empty, and close to the back. Below these again was a large soft mass filling the hypogastric and right inguinal regions, and stretching far towards the left. When the adhesions of this were detached, it was found to be the right ovary in a state of colloid degeneration. The uterus, which was found below this mass, appeared healthy on superficial examination. The left ovary had degenerated into a single cyst, filled with jelly, which not only occupied the upper portion of the pelvis, but sprang up as a globular tumor behind the uterus. The contents of the abdomen were incorporated into one tumor by this colloid material, and scattered through the gelatinous body could be found masses, varying in size, of the more fibrous or honeycomb-like material. It was a collection of these that were felt during life surrounding the uterus.

I present the specimens, but will not describe them minutely, both to save time, and because the general and microscopic appearance of this form of cancer has been so ably treated of by Sir James Paget in his Lectures on Surgical Pathology, to which I would refer you (p. 772). I would merely say that all the specimens I have examined of this disease correspond closely with his description, except, in two instances, the absence of the nucleated cells, which he (after Lebert) describes as lying free in the colloid substance.

The object which I had more particularly in view in bringing these specimens to your notice was to point out the great similarity of symptoms between this form of disease and that of multilocular ovarian tumor. In fact, so closely do they resemble each other that I believe it impossible to distinguish between them during life, except by tapping. This case was diagnosed as ovarian tumor by more than one able physician, and I certainly should have agreed with them before the tapping was performed, but, having met with these cases before in the practice of Dr. Washington

L. Atlee, I was familiar with the jelly-like matter, and so saved from an erroneous diagnosis.

Dr. Atlee, in his great experience of abdominal tumors, has met with but six of these cases, and they were so much alike that a description of one would apply to all, and in nearly all were they mistaken for ovarian disease by their physicians until the tapping revealed their true nature. This colloid or jelly-like material is unmistakable, and once seen can be always recognized. *June* 13*th*, 1878.

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11. *Case of recurrent fibroid of axilla.*

Presented by Dr. Nancrede, for Dr. J. Ashhurst, Jr.

The tumor was removed from Claude Conrad, aged 21 years, who was admitted, October 11th, 1876, to the University College Hospital for encephaloid of the axillary region, which diagnosis was revised to sarcoma. Professor Agnew removed the growth on the above-mentioned date, and the patient was discharged, cured, October 31st, 1876. Since that time five other small recurrent tumors have been removed by his family physician in Factoryville. On May 28th, 1878, Dr. J. Ashhurst, Jr., removed the present tumor, with two smaller growths, making in all *eight* tumors removed since the first was excised by Dr. Agnew. Macroscopically, this growth resembles strongly a uterine fibroid, but microscopically was seen to consist of numerous cells of a connective-tissue type, imbedded in a hyaline intercellular substance, presenting no appearance of fibrillation. Some of the cells were becoming decidedly spindle-shaped, evidently developing into fibre-cells. *June* 13*th*, 1878.

IX.—MISCELLANEOUS.

1. *The causal lesions of puerperal eclampsia.*

By James Tyson, M.D.

The writer would state at the outset that the time allowed for the preparation of this paper, had it included a much longer period, would have been altogether insufficient to have enabled him to base the conclusions arrived at upon personal observation. Cases of puerperal convulsions are fortunately not common, those which terminate fatally are less frequent, and *post-mortem* examinations still more rare. Of those which recover, very many occur under such circumstances that a previous clinical study is impossible. Thus, of five cases of puerperal eclampsia which have come under his observation in a practice of fifteen years, it so happened that none was seen until she was in the throes of labor. So that an examination of the urine in advance was impossible. All were primipara. In two of them, the subjects of which recovered, the convulsions did not occur until after the birth of the child, and did not reappear after a copious bleeding. They occurred very early in his practice, and no examination of the urine drawn by a catheter, and therefore separate from the lochial discharges, was made. They are therefore valueless. The third case, which again was not seen until convulsions set in, in what appeared to be the eighth month of pregnancy, occurred comparatively recently. The history developed the fact that the patient was enormously œdematous for some time before the convulsions occurred. The water drawn off by the catheter was highly albuminous, and contained granular and blood casts. She was not bled. Premature labor was induced, and she died about twenty-four hours after delivery. No *autopsy* was made, but the case was undoubtedly one of Bright's disease, and might be legitimately used in drawing conclusions.

In the fourth case of eclampsia, the urine was not examined until convulsions set in, but when examined immediately after the first convulsion, it was albuminous. In the first drawn, no tube-casts were found, but in the second and subsequent quantities, albumen, blood-disks, and tube-casts were present. These diminished gradually, and subsequently disappeared, the patient recovering completely. There was no œdema.

The specimens from the fifth case are now presented to the Society.

The contents of the larger four-ounce bottle, evidently bloody and highly albuminous, were drawn by a catheter after five convulsions had occurred, and *before* labor had set in. It represents twelve hours' secretion, and contains numerous highly granular casts, stained with hæmatin, blood-casts, epithelial casts, and blood-corpuscles.

The smaller bottle contains a portion of four ounces, drawn after she had been bled twelve ounces, and had received chloral, potassium bromide, a purgative dose of calomel, and an injection; had fallen into labor, and been rapidly delivered. Immediately after labor, she had received one-fourth of a grain of pilocarpin hypodermically, and fifteen minutes later was in a profuse perspiration and salivation, intelligence increasing momentarily. All this transpired in three hours after the first urine was drawn, and the four ounces, therefore, represent three hours' secretion. It is evidently lighter hued than the first specimen, but still contains considerable blood, is also highly albuminous, and contains large numbers of hæmatin-stained granular casts, blood-casts, and blood-disks. In both of the latter cases there was evidently Bright's disease.

This paper must consist, therefore, chiefly in an analysis of the work of others on the subject, and deductions from such examinations; and the only qualifications which the writer may be said to possess for such a task is the interest he has hitherto shown in renal diseases, in a somewhat close study of their symptoms, and in the examination of urine derived from them.

In the collection of material upon this subject, it has seemed convenient to arrange it under one of the following heads:

I. Writers making the causal lesions of puerperal convulsions, in the main, identical with those of Bright's disease, in most cases that form known as parenchymatous nephritis (*syn.*, acute catarrhal or desquamatous nephritis, etc.), of which the typical example is the acute nephritis concurrent or sequel to scarlet fever.

II. Writers whose observations and belief tend to controvert the view that puerperal eclampsia is due to Bright's disease.

III. Writers who admit that puerperal eclampsia is sometimes due to Bright's disease, developed as a consequence of pregnancy, but who also believe that it occurs as often independently of such cause.

I. *Writers who consider the causal lesions of puerperal eclampsia to be, in the main, identical with those of Bright's disease.*

Cazeaux says that, with the exception of six or seven cases mentioned by MM. Depaul and Mascarel, the circumstance of the almost constant

presence of albumen in the urine of eclamptic women entirely escaped the notice of the older observers.

Rayer, in his *Traité des Maladies des Reins*, published in 1840, first announced that he had repeatedly observed his Nephrite albumeneuse in pregnant women. His pupil, Cohen, at his master's suggestion, wrote a thesis, in which he attributed puerperal albuminuria to nephritis. But although this relation seems to have been mentioned by Blackall and Tweedie in England,* Dr. John C. W. Lever is generally acknowledged to be the first in Great Britain, and, with the exceptions named, the first in the world, to point out the relation between albuminuria and puerperal convulsions. Dr. Lever reports in *Guy's Hospital Reports*, *Second Series*, 1842, fourteen cases, in ten of which the urine was examined: albumen was found present in greater or less quantity in nine cases. In the tenth case, which was fatal, and in which no albumen was found in the urine, the *post-mortem* examination revealed acute meningitis, with four or five drachms of milky fluid in the ventricles. The note describing the condition of the kidneys reads as follows: "The kidneys gave out much blood, but were not dark; their texture coarse and flabby." It is impossible to infer from this description whether they were diseased or not. In the remaining fatal case, in which the urine was highly albuminous before delivery, with a sp. gr. of 1010, but free from albumen after delivery, no *post-mortem* examination was permitted.

As to the cause of this condition, Dr. Lever is of the opinion "that the gravid condition of the uterus, by its pressure, prevents the return of the blood through the emulgent veins; and hence is the cause of the renal congestion, and the consequent albuminous condition of the urine." This opinion he considers supported by the fact "that the urine was found to be albuminous only in those women who were affected by or had premonitory symptoms of convulsions."

In 1846, Cohen and Delpeck published a memoir demonstrating the extreme frequency of albuminuria in pregnant women. The late Dr. Simpson, of Edinburgh, was one of the first to call attention to the relation of albuminuria to puerperal convulsions. In 1847 he detailed to the Edinburgh Obstetrical Society (*Edinburgh Monthly Med. Journal*, 1847, p. 288) "some cases illustrative of the effects of Bright's disease, as denoted by the appearance of albumen in the urine, under the action of heat and nitric acid," from which he drew the following conclusions:

* See a valuable paper by Dr. Samuel C. Busey, on the "Artificial Induction of Premature Labor in Uræmia," read before the Clinico-Pathological Society of Washington, D.C.

1. Albuminuria, when present during the last periods of pregnancy, denotes a great and marked tendency to puerperal convulsions.

2. Albuminuria, in the pregnant and puerperal state, sometimes gives rise to other and more anomalous derangements of the nervous system without proceeding to convulsions; and Dr. S. had especially observed states of local paralysis and neuralgia in the extremities, functional lesions of sight (amaurosis, etc.), and hearing; hemiplegia and paraplegia more or less fully developed.

3. Œdema of the face and hands (going on occasionally to general anasarca) is one of the most frequent results of albuminuria in the pregnant female.

4. The presence of this œdema (3), or of any of the lesions of the nervous system (2) with or without the œdema, should always make us suspect albuminuria; and if our suspicions are verified by the state of the urine, we should diligently guard by antiphlogistic means, etc., against the supervention of puerperal convulsions.

5. Albuminuria and its effects (1, 2, 3) are far more common in first than in later labors, and these constitute a disease which in general disappears entirely after delivery. But Dr. S. had seen one case commencing with slight blindness, but no œdema, and ending gradually in hemiplegia, where the palsy partially remained after delivery, and after the disappearance of the albuminuria. In another, amaurosis came on with delivery, and had been present for six months when Dr. S. first saw her. She had no œdema or other symptom of albuminuria except the amaurosis; but on testing the urine it was highly albuminous.

6. Albuminuria with convulsions, etc., occurring in any labor later than the first, generally results from fixed granular disease of the kidney, and does not disappear after delivery.

7. Perhaps in puerperal convulsions, etc., produced by albuminuria, the immediate pathological cause of the nervous lesions is some unascertained but poisoned state of the blood. Was there a morbid quantity of urea in the blood? In several specimens of the blood of patients suffering under severe puerperal convulsions, furnished by Dr. S. to Dr. Christison and Dr. Douglas Maclagan, these gentlemen had been unable to detect any traces of urea. Was the poisoning material caseine in morbid quantity or quality? The dependence makes this connection worthy perhaps of some inquiry.

8. In cases of severe puerperal convulsions, etc., from albuminuria, the renal secretion is in general greatly diminished, and Dr. S. had found active diuretics apparently of great use along with or after venesection,

antimony, etc., especially where the case was offering to become prolonged.

9. Sometimes hemiplegia supervened during pregnancy without albuminuria, but this form did not seem to interfere materially or very dangerously either with the pregnancy or labor, the disease running its own usual course. In one case Dr. S. had seen the patient gradually but imperfectly recover the use of the palsied arm after delivery. In another no improvement occurred.

Garrod (*London Lancet*, 1848) says the presence of albumen in the urine, independent of blood or pus, is owing to some morbid condition of the kidneys.

Devilliers and Regnault (*Arch. Gén. de Méd.*, t. xv., xvi. et xviii., 1848) verified the coincidence of eclampsia and albuminuria. They report eight autopsies of cases of puerperal eclampsia, in four of which a disease of the tissue of the kidney was found. This condition thus early they called *albuminous nephritis.*

Dr. Cormack (*Med. Times*, London, 1850) also pointed out the coincidence of eclampsia and albuminuria.

Dr. G. O. Reese (*London Medical Gazette*, 1851) says, "The presence of albumen in the urine must be regarded as significant. Continued albuminuria unconnected with lesion of the kidney is rare."

Frerichs (*Die Brightsche Nierenkrankheit und deren Behandlung*, 1851) attributed puerperal convulsions to Bright's disease, and was the first of the Germans to devote especial attention to the nephritis of pregnancy.

On p. 215 of the above-named work he says, "True eclampsia occurs only in pregnant women suffering with Bright's disease, and it bears to the latter the same causal relation as convulsion and coma to Bright's disease in general; it is the result of the uræmic intoxication, with which also in its mode of manifestation it agrees."

This, Bright's disease, he ascribes to two causes: 1st, the alteration of the blood crasis caused by pregnancy; and, 2d, the mechanical interference with the venous circulation in the abdomen. By *crasis* Frerichs seems to have meant an impoverishment of the material blood consequent on the drain of nutritive matters for the nourishment of the fœtus.

Carl Braun, in January of the same year (1851), declared that, "As a rule, *eclampsia vera puerperalis* is found intimately connected with *diabetes albuminosus*" (*Ueber Eclampsie und Albuminurie in der Zeitsch. d. G. d. Wiener Aertzte*, 1851, Bd. i., S. 57). And in his *Lehrbuch der Geburtshülfe*, etc., Vienna, 1857, he devotes a chapter to the subject, which was shortly afterwards translated into English by J. Matthews Duncan, of

Edinburgh, and is well known to the profession as *Braun on the Uræmic Convulsions of Pregnancy, Parturition, and Child-Bed.*

In this volume he says, "The coincidence of eclampsia and albuminuria is an undisputed fact, which is verified by the very numerous observations of Lever, Simpson, Devilliers, Regnault, Dubois, Danyau, Cazeaux, Cormack, Blot, Helft, Frerichs, Litzmann, the author, and many others, as also by daily observations. This has opened a new path to the knowledge and treatment of this most dangerous disease, so that the eclamptic convulsions of women during pregnancy must be considered to be identical with the fits of adults in general that are produced by uræmia in the course of acute Bright's disease. This I publicly declared in January, 1851, and at the same time Frerichs published his most convincing classical treatise on the subject. It must now be considered an axiom in theory as well as in practice."

Braun proceeds to prove his proposition by a long argument, from which I abstract only such as is based upon *post-mortem* examinations or the presence of albumen in the urine. Of forty-five cases published by him, fifteen died, but *post-mortem* examinations were made only in twelve. In seven of these Bright's disease was proven to be present by microscopic examinations, made by Wedl and others. In all the remaining cases the kidneys were *hyperæmic;* but from accidental obstacles no microscopic examination was made. But, as he says, "this cannot be considered any proof of the absence of Bright's renal exudation." On the other hand, the hyperæmia would seem to afford presumptive evidence of the presence of Bright's disease. Gustav Braun further communicated to him six cases with fatal termination, in three of which Bright's disease was demonstrated beyond a doubt. He further alludes to one case published by Lumpe, two by Hecker, four by Devilliers and Regnault, three by Simpson, two each by Sabatier and Hohl, one by Blot, Cohen, Wieger, Litzmann, and Crede, in all of which were found Bright's disease of the kidney after death. Hasse never saw eclampsia puerperalis without Bright's disease. Braun concludes with the statement, "There are above thirty cases known to me offering positive proofs of the intimate connection between Bright's disease and eclampsia, and they are far too numerous to admit of the opinion that there is only an accidental connection between the two being still entertained. Their value as arguments is very much enhanced by the circumstance, that in negative observations the histology of the kidneys was examined microscopically only in very rare instances."

He further states that in an observation by Oppolzer and himself, they prognosticated the outbreak of eclampsia two days before its occurrence

from the presence of abundant albuminuria, and from the blood containing a large quantity of urea. Also "Devilliers, Regnault, the author (*i.e.*, Braun), Litzmann, and Wieger have published a complete series of observations, in which albuminuria and exudation clots (casts?) occur along with Bright's disease during pregnancy, lead to spontaneous premature labor under different uræmic phenomena, and then terminate in speedy recovery, having no further injurious consequences, and producing no eclampsia. The circumstance that eclampsia does not occur in every case of Bright's disease during pregnancy, as Litzmann has very correctly remarked, can be accounted for by this result following only when the blood has been very considerably impregnated with the excrementitial elements of the urine, which always implies a profound, or at least extensive disease of the renal tissue." Alluding to the large amount of albumen and the large number of exudation casts covered with abraded glandular epithelium, already in part undergoing fatty degeneration, or even passing into detritus, "no one can really believe," he says, "as Litzmann has strikingly pointed out, that a disease in the kidneys furnishing such products has just been developed within the last few hours."

As to the causes of Bright's disease, Braun includes, among the more remote, the peculiar changes in the blood of pregnant women, the pressure of the pregnant uterus, and the stoppage of the venous blood in the kidneys caused thereby. Of cases of eclampsia, eighty per cent. occur in first pregnancies, in which, on account of the greater resistance of the abdominal walls, a powerful counter-pressure on the kidneys is generally produced. In cases of repeated pregnancy, the pressure connected with a plural pregnancy, with deformed pelvis, hydramnos, the large size of the fœtus, and a high position of the womb are frequently met with where eclampsia occurs. He says, further, that the cases in which albuminuria occurs during the first half of pregnancy cannot be referred to pressure on the renal veins, but may, according to Litzmann, be traced back to catarrhal irritation of the urinary passages, or, in cases of really existing Bright's disease, to a complication existing before the pregnancy. Neither of these two observers appear to have met puerperal eclampsia as early as has been observed by more recent observers, during the second month. Finally, he admits that "congestion of the venous blood in the kidney is not the only cause of Bright's disease; for among cases of eclampsia there are twenty per cent. of premature births; and abortions, even in the fifth and sixth months of pregnancy, having fatal terminations, have been observed by Velpeau, Harris, Pätsch, and others. It is reserved for future inquiries to answer the question, whether or not eclampsia of the first half of pregnancy

appears always contemporaneously with Bright's disease." The fact that after the womb is evacuated, and at the same time the impediment to the free circulation of venous blood is removed, the albuminuria disappears with great rapidity is pointed out as showing the intimate connection between the two, that is, the pressure and Bright's disease.

The name of Litzmann is conspicuous in the history of the relations of puerperal eclampsia to Bright's disease. He early entered the lists in support of the dependence of the former upon the latter. In his first paper (*Deutsche Klinik*, 1852, No. 19, *et seq.*) he reports, first, six cases of parturient women in which there were symptoms of Bright's disease, as shown by albuminuria, diminished secretion of urine, uræmia, in all of which, except one, eclampsia occurred. The latter was that of a woman in whom, in the first pregnancy, there were the symptoms of Bright's disease *with* convulsions. In her second pregnancy there were the same symptoms, including albuminuria, uræmic intoxication, and amaurosis, but without convulsions; the albuminuria disappearing after delivery. In a second series of six cases there were the same symptoms of Bright's disease without eclampsia. One of these cases died, revealing evident Bright's disease of both kidneys.

Litzmann agrees with Rayer and Lever as to the cause of the albuminuria and Bright's disease of pregnancy. He attributes them to mechanical pressure and to stagnation of circulation, especially in the kidneys. He observed the development of albuminuria most commonly in primipara (in which the abdominal muscles do not yield so easily to pressure as in the multipara), or in multipara with narrow pelves, or in twin pregnancies, or a very large child, or hydramnos. In three cases of albuminuria the women were affected with a chronic catarrh of the lungs; the impaired pulmonary circulation might have given rise to a venous hyperæmia of the kidneys. The frequent instantaneous disappearance of the albuminuria with the emptying of the uterus, which was pointed out by Rayer, is also in favor of the theory of mechanical pressure.

Litzmann believes with Lever, Devilliers, and Regnault that the usual existing causes of Bright's disease in the non-pregnant, such as cold and excess in alcoholic drinks, cannot operate in producing the Bright's disease of pregnancy. He also does not believe that mental distress and sorrow can favor the development of the disease.

The lesion of the kidneys found by Litzmann corresponded mostly with those of an early stage of Bright's disease. In his experience, Bright's disease never develops itself in the pregnant woman before the eighth month; but he alludes to two cases observed by Devilliers and Regnault, where it

began in the sixth month, and produced eclampsia. This, however, he considers the earliest possible period, as before the sixth month the uterus is hardly large enough to exercise any pressure on the renal veins, and where albuminuria exists earlier, he attributes it to a catarrhal irritation of the ureters and bladder with purulent secretion, which he was the first to describe, or to chronic Bright's disease existing before the pregnancy.

For the diagnosis of this affection, he considers the only reliable signs to be albuminuria and tube-casts. Œdema is often wanting. He also accepts Frerichs's view, now very generally given up, that it is not the retention of urea in the blood which causes the eclampsia, but carbonate of ammonium, derived from a decomposition of the urea. Litzmann considers that his observations confirm this theory.*

In a second paper, published also in the *Deutsche Klinik*, 1855, Nos. 29 and 30, Litzmann's object is to support the views advocated by him in 1852 against the attacks of Scanzoni and others. More cases are reported, including those of Simpson, Hecker, Lumpe, and Braun, already given in the abstract of Braun's views. Also another by Litzmann himself, in which the first pregnancy was attended by eclampsia; the second and third were normal; fourth, eclampsia; fifth, no eclampsia, but amaurosis; sixth, eclampsia at sixth month; seventh, eclampsia; eighth, abortion at second month; ninth, pregnancy terminated safely, although Bright's disease was evidently present, the urine being loaded with albumen and containing tube-casts. All the pregnancies were accompanied by œdema and temporary suppression of urine. Perspiration, however, was increased at these times. The inference of Litzmann was that a Bright's disease started during pregnancy may often continue as a chronic disease after delivery.

E. Robins (*London Lancet*, 1852) says, "Albumen appears in the urine during gestation where sufficiently advanced to occasion a habitual congestion of the kidneys."

In 1856, Imbert-Goubeyre published his thesis *De l'Albuminurie Puerpérale et de ses Rapports avec l'Eclampsia* (Mémoires de l'Académie Impériale de Médecine, Paris†), crowned by the French Academy, in which he says, "It is at present no longer possible to deny the connection

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* It may be remarked here that this theory of Frerichs's has been again brought forward by Spiegelberg (*London Lancet*, December, 1870), founded upon the result of a series of experiments conducted by himself and Heidenhain.

† Although this is the date of publication of Imbert-Goubeyre's paper, it was probably written between 1850 and 1855, as it is quoted freely as a "Manuscript Memoir" by Cazeaux, in the fifth edition of his Midwifery, the preface of which is dated November, 1855.

of eclampsia with albuminuria, or Bright's disease. Taking all facts together, true eclampsia is nothing but Bright's puerperal disease in which convulsions prevail: it is Bright's disease accompanying pregnancy, and it predominates then with a particular form of cerebral accident, which is eclampsia. This is proved by its symptomatology, its progress, its termination, its prognosis, and its anatomical lesions."

Cazeaux early examined the kidneys from fatal cases of puerperal eclampsia with Rayer, but the earliest publication of his views which I find is in the second American from the fifth French edition of his Midwifery. The latter was issued in 1855.

He says at the outset (p. 289 of the American edition), that as albuminuria is generally the symptom of an organic disease of the kidneys which almost always proves fatal, it becomes important to determine whether its presence in pregnancy is due to the same cause, or whether it is merely one of the numerous modifications produced in the economy by gestation; for, in the first place, it is a serious condition, and in the second, a temporary functional disorder which will most probably disappear with the cause.

In answer, he says, "Observation shows that in almost all the cases in which women die of the convulsions which too frequently complicate albuminuria, the kidneys present the anatomical characteristics of albuminous nephritis, the more or less advanced degrees of alteration appearing to correspond with the duration of the disease and the amount of albumen discharged. Many times have I had occasion to observe this fact, and fearing lest I should interpret the alterations erroneously, have almost uniformly presented the kidneys to the examination of M. Rayer, who generally recognized in them the second, sometimes the third, and only once the fourth degree of alteration."

"The learned physician of La Charité considers the more frequent occurrence of the anatomo-pathological characters of the second degree of the disease to be due solely to the recency of the latter, and by no means to a difference of nature. It is no less the consequence of a renal hyperæmia, which he supposes may be caused in many cases by compression of the emulgent veins by the enlarged uterus, and the consequent obstruction to the return of venous blood. That, in simple cases, it generally disappears promptly after delivery is probably due to the consequent cessation of the congestion of the kidney, which was maintained by the pregnancy."

Cazeaux says, further, that many of the objections to this view may be met by the fact first observed by M. Pidoux, and too often ignored, "That the secretion of urine is not confined to the kidney, since it takes place

previous to the formation of the latter. The process of assimilation, which is so active in the fœtus, can only be understood by supposing a contemporaneous process of decomposition. The blood which flows to the organ is already charged with the elements of urine, which are to be separated from it in the passage. The function begins in all parts of the economy by this admixture of heterogeneous elements with the blood, and is completed in the kidney by their elimination from the circulating fluid, which is returned in a purified condition. . . . To study the latter organ exclusively, when we wish to obtain a physiological idea of the function, is to neglect an important element; so, also, always to expect to find the cause of the disorders of the urinary secretion in alterations of the kidney is to overlook a multitude of other causes which have a corresponding influence. The elements of the blood conveyed by the renal artery exist in health in a fixed proportion, and certain of them are destined to be eliminated by the kidneys. Now, it is easy to understand that if an alteration in the structure of these organs is capable of modifying both the quantity and quality of the matters eliminated, an alteration of the fluid, such, for example, as the diminution or increase of its solid or fluid parts, may have the same effect. Clinical observation and *post-mortem* examination give constant support to this idea; for, though we sometimes find a material lesion of the kidney to which we attribute the albuminuria, we are very frequently obliged to recognize it also in certain general diseases, as scarlatina, typhoid fever, cholera, etc., in which the renal affection is often wanting. In Bright's disease itself, or at least in certain cases in which the presence of albumen in the urine, and all the other symptoms of granular nephritis, had been detected during life, the *autopsy*, though conducted with the greatest care, revealed no alteration of the kidneys (Forget, of Strasbourg). Finally, Kennedy found the kidneys to be perfectly healthy in several cases of dropsy with albuminuria following scarlatina. Must we conclude from these that Bright's disease did not exist in those cases? Certainly not; the disease was present, but the pathological alteration was confined to the fluids."

"This," he continues, "may be the case in pregnancy." "The composition of the blood is clearly altered. It contains less albumen, fewer corpuscles, more fibrin, and, according to some, also more urea. Its influence on the kidneys cannot be denied, varying with the duration and degree of alteration. If the latter be slight, the effect will be almost nothing; if more prolonged or intense, it will cause greater derangement of function, indicating its existence by albuminuria and anasarca. All this may take place *without any appreciable lesion of the kidneys.*"

He considers the mistake of M. Blot and others who insist so strongly upon the cases of puerperal albuminuria without renal alteration, with the object of proving that the presence of albumen is but very rarely the consequence of Bright's disease, consists in their confining Bright's disease to the renal alteration exclusively. We may have *variolæ sine variolis*, —typhoid fever without the intestinal eruption; why not Bright's disease without renal alteration?

Such is the argument of Cazeaux with regard to the relation of albuminuria and pregnancy. As to the relation of albuminuria to eclampsia, he says first (p. 716, *op. cit.*), "Since the presence of albumen is discovered almost constantly in cases of eclampsia, the severest mind can hardly avoid establishing a more or less intimate relation of causality between the two facts; . . . and since albuminuria is present in the immense majority of eclamptic women, it, or rather the disease of which it is a symptom, may be rightfully regarded as the predisposing cause of eclamptic convulsions."

The fact that the amount of albumen is markedly increased during the eclamptic fit and generally diminishes after it, which is used by some to prove that albuminuria is the result of eclampsia rather than the cause of it, he disposes of practically by denying the possibility of eclampsia occurring without being preceded by albuminuria. He says, "I can understand why there might be hesitation in regard to this point, if a single case could be cited in which it had been proved that the urine was entirely free from albumen for several weeks before the appearance of the accident. This, I believe, has never been done, but often, on the other hand, albuminuria has been known to be present for some time before the convulsions occurred." It is easy to understand, also, how a renal congestion may take place in the kidneys in common with other internal organs during labor, and the increased secretion of albumen should result from such congestion. He admits also that the condition of the blood which results from a long-continued albuminuria, by giving rise to a peculiar excitement of the cerebro-spinal centre, may itself become the direct cause of the convulsion, or, at least, which is more frequently the case, render it more susceptible to the excitements which reach it either from without or from previously irritated internal organs.

In speaking of the pathological anatomy of the disease, he says, "What we have stated in regard to the almost uniform coincidence of albuminuria with eclampsia, and to its common connection with lesions of the kidneys, sufficiently indicates that the anatomical lesions are hereafter to be sought in those organs. For our own part we have never failed to do so for the

past ten years, nor do we hesitate at the present time to consider albuminous nephritis as one of the most common lesions after puerperal convulsions. As already stated, the kidneys have almost universally presented the anatomical characters of nephritis, the more or less advanced degrees of which appeared to coincide with the chronicity and abundance of the albuminuria.

"Other observers, amongst whom I may mention MM. Blot and Depaul, state that usually they have met with no disease of the kidney, and, regarding the above-mentioned facts as altogether exceptional, insist that in the majority of cases Bright's disease has no connection with eclampsia.

"In the first place, I would call attention to the fact that I do not regard Bright's disease as residing in the lesion of the kidneys exclusively (page 294), and although the kidneys should present nothing abnormal, the alteration of the urine is sufficient to prove its existence. I might, therefore, strictly pay no regard to the facts mentioned by my opponents; but let us examine whether, independently of the opinion which I support, the observations of MM. Blot and Depaul are of much value. They have found nothing, say they; but perhaps their not having done so is their own fault in not having examined sufficiently, and I have to acknowledge that hitherto I had committed the same error. Works recently published in Germany show, in fact, that the naked eye is entirely incompetent to detect anatomically the commencement of albuminous nephritis, and that the first degrees of renal alteration can be discovered only by the microscope."

W. R. Basham, M.D., in his book on Dropsy (1858), says that the albuminuria in pregnancy is due to the obstruction of the circulation in the kidneys caused by direct pressure of the gravid uterus. The albuminuria may pass away after delivery, but sometimes, if the pressure is prolonged and excessive, it leads to true kidney-disease, in which case there are also found tube-casts.

Dr. Krassing in *Spitals Zeitung*, 1859, reports nineteen cases of eclampsia which occurred in the clinic of Braun, in Vienna, in 1857 and 1858. Nine died. In all the nineteen cases save one there was reason for believing that Bright's disease was present. In one case with sixty paroxysms, no albumen was found in the urine. In ten cases there was œdema. In one case where eighty-one attacks were observed there was no œdema. Of the cases examined after death, there was found hyperæmia of the kidney with inflammatory exudation in three cases, fatty degeneration in four cases, atrophy in one case, and no morbid change in one case.

Dr. Perrie (*Dubl. Qu. of Med. Sci.*, vol. xxix, 1860) says albuminuria is most probably caused by renal congestion, the effect of direct pressure.

Dr. L. X. Bourgeois, in a memoir (*De l'Influence des Maladies de la Femme pendent la Grossesse sur la Constitution et la Santé de l'Enfant*) read before the Imperial Academy of Medicine, in 1861, says puerperal eclampsia is due to Bright's disease.

Dr. Bosse (*Spitals Zeitung*, 1862) relates fourteen cases of eclampsia which occurred within thirty-one months among four thousand five hundred women at the Lying-In Hospital at Gratz. Of these fourteen cases, thirteen were primipara; eight recovered and six died. In *all* these last there was intense Bright's disease, and in five cases œdema of the lungs.

Bedford (*Principles and Practice of Obstetrics*, 1861, p. 506) says, "Albuminuria is due to the following causes: 1st, a change in the composition of the blood; 2d, a change in the kidneys, either structural or dynamic; 3d, pressure of renal veins."

The late Prof. George T. Elliot (*Obstetric Clinic*, 1868) reports a large number of cases of puerperal albuminuria and eclampsia, in many of the fatal cases of which *post-mortem* examinations revealed lesions of the kidneys; and although he does not state explicitly his views with regard to their necessary relationship, it is evident he inclines to this view. His colleague, Prof. Barker, however, says, on p. 104 of *The Puerperal Diseases*, "Another would only make two classes, the uræmic and hysterical. My friend and colleague, Prof. Elliot, followed this last division, calling those convulsions which were general in their character, and attended with loss of consciousness, eclampsia; and he believes that these were always associated with renal lesions."

Bailey (*Dictionnaire de Méd. et de Chirurgie*, "Eclampsia," 1870) says, "The only organic lesion in eclampsia is that of the kidneys; it consists of a congestion, of desquamation of the epithelium of the tubules, fibrinous exudation into the tubules and into the parenchyma of the kidneys, fatty degeneration of the elements, and, in rare cases, glandular atrophy, which forms the highest anatomical expression of a chronic form of Bright's disease."

M. Moss (*Prog. Méd.*, 1873) found the kidneys, in a case of eclampsia, to present the characters of *parenchymatous nephritis.*

Leishmann (*System of Midwifery*, 1873, p. 475) says, "With rare exceptions, then, arising from these or similar causes (epilepsy, diseased conditions of nerve-centres, and hysteria), puerperal eclampsia may be looked upon as essentially connected with uræmic poisoning, which, again,

is associated with or dependent upon an albuminous condition of the urine." And as to the cause of the renal lesions, he believes (p. 254) that it is due to the pressure of the gravid uterus in many instances, while he is inclined to think that this explanation has been too readily accepted the solution of the case, especially in cases where albuminuria has appeared so early that pressure is impossible. Without pretending to account for the condition in these cases, he quotes Dr. Tyler Smith thus: "In such cases," says Dr. Smith, "the disease appears to me to depend upon reflex irritation of the kidneys by the gravid uterus, similar to the irritation of the salivary glands, the mammæ, thyroid, etc., and not upon mere pressure alone."

Dr. Depaul (*Archives de Tocologie*, 1874) reports several cases of eclampsia in the sixth month of gestation, with symptoms of uræmic poisoning and albuminuria, and total absence of œdema.

Dr. Johnson (*Lectures on Bright's Disease*, 1874, p. 118) admits that puerperal eclampsia may occur without albuminuria, and, on the other hand, albuminuria in pregnant women may be unassociated with convulsions. He limits his discussion, however, only to puerperal albuminuria, and, referring to illustrative cases, he arranges the latter into four classes:

1. Cases of women suffering from chronic Bright's disease, who passed through all the stages of parturition many times *without eclampsia;* free secretion of urine continuing, and preventing it by maintaining elimination.

2. Cases (mostly primiparæ) where the *pressure of the gravid* uterus on the vena cava produced a passive renal congestion, albuminuria, and uræmic *convulsions.* He argues that in multiparæ the abdominal walls are more yielding, and pressure is thus avoided.

3. Cases of *acute desquamatic nephritis*, originating during pregnancy from exposure to cold and wet, drinking, or from zymotic blood-poison, or which may be due to some blood-change, to the evolution of the uterus and the growth of the fœtus. The renal symptoms may pass away after delivery; they may or may not return with the next pregnancy. In cases where the albuminuria is persistent, ultimately uræmic symptoms occur, and after death the kidneys are found either *contracted and granular* or *large and pale.*

4. Cases in which albuminuria and convulsions appeared for the first time *after delivery.* The urine is in such cases indicative also of an *acute desquamatic nephritis;* these cases are pathologically allied to, and sometimes associated with, a form of septicæmic puerperal fever. The

cause of the renal symptoms here is "the blood-contamination consequent on the absorption of morbid materials from the interior of the uterus after parturition." The renal symptoms may pass away, or the disease may become chronic and terminate in a *large white kidney*.

Dr. M. Peter, in a series of lectures on puerperal eclampsia, their causes, nature, and treatment, published in the *Archives de Tocologie* for 1875, first described, not very precisely, the *post-mortem* appearances of the kidneys of two primiparæ dying in puerperal convulsions. The organs were somewhat enlarged and very anæmic; the cortical substance presented the appearance of cooked veal, and the pyramids were hyperæmic. In the second case, in addition to the above, he says the kidney corresponded anatomically with the third form of Bright's disease, described by Rayer. The injection at certain points was so great that the veins were distended with blood-clots. There was also albuminuria, and the microscope revealed numerous blood-globules, epithelium desquamated from the uriniferous tubules, and granular casts. "In brief, then," he says, "our eclamptic had had Bright's disease of puerperal origin."

The next lecture he begins by saying, "All pregnant women attacked with eclampsia are albuminuric. Such is the proposition formulated by Cazeaux, confirmed by Frerichs, by a professor in Vienna, Braun, and by all those who have studied eclampsia in the pregnant woman. . . . To this proposition (and for the great glory of a certain theory) some have opposed this argument, that there are pregnant women attacked with eclampsia who are not albuminuric. And they have cited in favor of this opinion as many as *six* cases,—*six cases* against the hundreds of cases of albuminuric eclampsia!"

Doubtless, he admits, there are exceptions, but these cannot change the rule, and it must be remembered that a large number of pregnant women having albuminuria may lose it temporarily. This is beautifully shown by Bailey in his article on "Eclampsia," in *Dictionnaire de Médecine et de Chirurgie Pratique*. He has observed that not rarely albuminuria in pregnant women disappears for several hours, but subsequently reappears again; and the cause of this may be attributed to a congestion which may suddenly vary in degree or disappear. The six cases in question he thinks cannot be explained otherwise than that the examination was made during the short period when the urine ceased to be albuminous. "I insist," he says, "upon my original proposition,—'all pregnant women attacked with eclampsia are albuminuric.'"*

* Dr. Peter prefers, not inaccurately, to speak of the passage of albumen into the urine as a "transudation of serum," rather than of albumen from the blood.

He says further that an analysis of the urine shows, as we would expect, a greatly increased elimination of urea during the pregnant state. The analyses of Quinquaed have shown that the pregnant woman, instead of secreting 22 to 24 grammes of urea in the twenty-four hours, eliminates 30 to 38 grammes,—nearly twice as much. This uropoiesis for two of course necessitates the movement of an increased amount of blood through the kidney, and produces a functional hyperæmia and a consequently increased intra-vascular pressure, which facilitates what he calls not merely an albuminuria, but a *serumuria*. Further, by the microscope we are enabled to judge of the exact condition of the kidney. The presence of renal epithelium of granular and hyaline casts shows that in the situation where the desquamation has taken place the kidney has absolutely lost the power of secretion: it has become a mere passive organ, through which the serum filters as it would through filter-paper. Hence the integrity of the kidney may be said to be inversely proportional to the quantity of albumen. With such defective secreting power, the urea must accumulate in the blood. But there is not only an accumulation of the urea, but also of all the constituents of the urine. Analyses of the blood of eclamptic patients have shown three times the quantity of urea found in normal blood. But the extractives have also been found increased threefold; so with the creatinin; and we should not therefore speak of an uræmia so much as of an urinæmia. Such a condition Dr. Peter likes to speak of as an *autotyphization* of the blood, or of a *typhization urinemique*.

Dr. Peter proceeds further to fortify his views as to the pathology of puerperal convulsions by the observations, familiar to most of us, of Dr. A. Mahomed, made in the Fever Hospital of London, and published in the *Transactions of the Royal Medico-Chirurgical Society of London* for 1874. These were to show that there was a like increased arterial tension in the vascular system of the pregnant woman, and in what he calls the pre-albuminuric stage of Bright's disease, during which the crystallizable principles of the blood transude into the uriniferous tubules before the albumen. Such transudation Dr. Mahomed proved by the guaiacum test,—the tincture of guaiacum striking with urine under these circumstances the characteristic delicate blue color of the coloring-matters of the blood,—while there is still no albuminuria. The increased tension was shown by sphygmographic tracings. The same condition is proven by the miliary aneurisms observed by ophthalmologists and the hemorrhages into the eyeball which have occurred under these circumstances. Finally, he considers them further sustained by the peculiar views of the physiologist Küss on the secretion

of urine, who believes that in health the serum of the blood, with its contained albumen and salts, is transuded into the Malpighian capsules, and that the albumen is reabsorbed by the epithelium of the convoluted tubules.

Those who claim that the phenomena of puerperal eclampsia are due to deranged circulation, the result of pressure upon the renal veins and inferior cava, are asked to account for the convulsions which present themselves in the early months of pregnancy, before the uterus has risen above the cavity of the pelvis; while, unfortunately for this theory, we see women who have enormous cysts of the ovary who have neither eclampsia nor albuminuria; others with enormous fibromata of the uterus without any trouble with the renal secretion. The pressure, according to Dr. Peter, is within the blood-vessels of the kidney, and not without. Dr. Peter, moreover, does not forget to say that not all albuminuric women are necessarily attacked with eclampsia.

Dr. Bourneville, in the *Archives de Tocologie*, 1875, p. 194, reports four cases of puerperal eclampsia, in all of which albuminuria was present. Two terminated fatally, but a detailed record of one case only is furnished. She was a primipara; the urine was loaded with albumen, and contained granular tube-casts and red blood-disks. Convulsions set in during labor. The *autopsy* revealed incipient putrefaction on the anterior parts of the kidneys. In other places the tissue of the organ was pale, except the cortical substance, which was yellowish in hue. There was no notable injection, except a little black blood in the larger vessels. The description lacks preciseness, but associated with the albuminuria Bright's disease may be inferred.

Dr. Eugène Marchal (*Archives de Tocologie*, 1875, p. 690) reports three cases of puerperal eclampsia which terminated in death: 1*st case*.—Multipara: urine dark-colored, highly albuminous, and the microscope revealed presence of blood. *Autopsy*.—Kidneys hyperæmic; hypertrophy of the epithelium of the tubules, with beginning fatty infiltration; no alteration in the connective tissue, nor any deformation in the tubules (first stage of Bright's disease).

2*d case*.—Primipara: unmarried; albuminuria. *Autopsy*.—Kidneys highly congested, chiefly in the cortical substance; the epithelium of the tubules hypertrophied and fattily infiltrated (latter stage of Bright's disease).

3*d case*.—Primipara: unmarried; delivery normal of healthy child; *no albumen in urine before delivery*, but as soon as eclampsia set in *albumen* was found in the urine copiously. Twenty-four hours later the patient died (having had twenty-two attacks). *Autopsy*.—The kidneys

strongly hyperæmic; the epithelium of the tubules hypertrophied, but no alteration in the connective tissue, and no change in the shape of the tubules. Liver fatty.

Bartels, in his article on the "Acute Parenchymatous Nephritis of Pregnancy," in Ziemssen's *Cyclopædia of Medicine*, 1877, says, "Apart from hemorrhagic extravasations,—which are wanting, too, in a great many cases of scarlatinal nephritis,—the pathological changes in the renal disease of pregnancy are identical in every respect with those of the other forms of acute nephritis." Also, "When we study carefully the descriptions of the microscopic appearances of the kidneys removed from the bodies of lying-in women who have died of eclampsia, a number of which were published by Litzmann in the *Deutsche Klinik* for 1855, we find that they correspond exactly with the appearances which, as we saw in the preceding chapter, are presented by the acute renal inflammations due to other causes; for instance, by scarlatinal nephritis that has run a protracted course. The kidneys are larger and heavier than normal, from thickening of the cortical substance. The cortex is anæmic, of a pale yellowish color, and of a swashy consistency. What resemblance is there here to the very vascular, dark-colored, and usually firm and compact kidneys that we find where venous stasis has existed?* It is unnecessary to add anything concerning the changes in the other organs of the body that are found at the *autopsy*. I will only mention that dropsical effusions are seldom altogether absent. Thus, Braun found œdema in thirty-nine out of forty-four cases of eclampsia."

On p. 313 of the same article, Bartels also says, "That acute uræmia occurs more frequently in the nephritis of pregnancy than in the other forms of this renal disease will be admitted even by those who, like myself, are not inclined to regard every case of eclampsia or mania occurring during parturition as of uræmic origin, even although the urine, after the outbreak of the symptoms, contains some albumen."

Bartels contends strongly against the theory of renal pressure adduced by Lever and Rosenstein as causes of the nephritis. He says, "It must at once strike the student that not a single iota of evidence founded upon fact can be adduced to establish this altogether gratuitous assumption of a compression of the renal veins by the pregnant uterus. The clinical symptoms are by no means the same as those of obstructive engorgement, and the anatomical appearances not only do not resemble those of cyanotic induration of the kidneys, but correspond in every particular with those of

* Interstitial nephritis is here alluded to.

parenchymatous nephritis." He further says that the position of the renal veins protects them, under all circumstances, from any direct pressure on the part of the pregnant uterus, and any one who has had the opportunity of opening the body of a woman far advanced in pregnancy, and has observed the relative position of the abdominal organs, could not fail to perceive how inadmissible is the theory that a mechanical pressure is exerted upon the renal veins by a distended uterus. He then proceeds to demonstrate the anatomical impossibility of such pressure occurring, for which reference must be made to his article.

With regard to the actual cause of the parenchymatous nephritis, except to say that there must be some special cause in the pregnancy itself, he does not bring forward any, but says that, "For the present there is nothing left for us but to record the fact that parenchymatous inflammations of the kidneys and liver (as originally shown by Virchow with regard to the latter organ) may be developed during pregnancy, and to confess that we do not know what causes them."

Dr. W. Howship Dickinson (*On the Pathology and Treatment of Albuminuria*, 2d ed., London, 1877, p. 158) says, "Another condition—acting probably mechanically, and producing, as does heart-disease, venous congestion of the kidney, and the train of organic changes and constitutional results which spring therefrom—is pregnancy.

"The disproportionate results, as compared with other forms of renal disease, particularly in respect to convulsions, which have been stated to occur in one-fourth the cases of albuminuria of pregnancy, may be perhaps due to the disturbing influence of labor, the exaltation of nervous susceptibility which would seem to accompany the act, or the effect which it has, whether by exhaustion or febrile action, of destroying the equilibrium of the nervous system. Thus, under the sudden influence of parturition, uræmic convulsions may result from an amount of uræmia which might otherwise be inadequate to produce them."

Julius Althaus, in his recent work on *Diseases of the Nervous System* (G. P. Putnam's Sons, New York, 1878), has an excellent article on puerperal eclampsia, in which he says, referring to the view of "Scanzoni and some other high obstetric authorities," who attribute the albuminuria of pregnancy to venous congestion, produced by an attack of eclampsia as the primary disease, "This view is most certainly not borne out by the facts of the case. In the first place, we have to notice the physiological fact that removal of both kidneys in animals gives rise to uræmia and convulsions. Secondly, it is contrary to fact that convulsive attacks tend to produce albuminuria, because even after a long series of epileptic attacks

no albumen is found in the urine. Thirdly, the disease is not mere albuminuria, or passive transudation of albumen from congestion, but real nephritis, as shown by the microscopic appearances of the urine during life, and of the kidneys after death. Fourthly, this nephritis is almost invariably prior to the convulsions; for although in some well-observed cases albumen has been absent from the urine before a fit, and present after one, it seems that œdema has always preceded the fit. Now, it is well known that albumen in the urine is not always the first symptom of nephritis: for in certain cases of scarlatina, dropsy has been observed before the albuminuria."

As to the cause of the nephritis before and after parturition, he says it is obscure, but brands the pressure theory as erroneous, for reasons practically the same as those already given; also, that in the absence of any better reason we may consider both the nephritis and eclampsia due to the circulation of some irritant material in the blood. He does not even admit the occasional operation of one of the usual causes of nephritis, as cold, excess in drinking, etc., for the reason that the disease generally vanishes after the lying-in period.

I have not had access to the work of Prof. Hervieux, physician to the Maternité Hospital, Paris, *On Puerperal Diseases*, but Prof. Barker in his own book on this subject says Prof. H. "seems to regard puerperal albuminuria as mainly caused by what he calls 'puerperal poison,' and as analogous to the albuminuria which occurs from the scarlatinal poison."

From the standpoint of pathological anatomy, Rindfleisch, in his *Pathological Histology* (1875), treating of acute parenchymatous nephritis, mentions "puerperal processes" as one of its causes.

Cornil and Ranvier (*Pathological Histology*, 1876, p. 1035) say, "Nephritis with albuminuria being more or less persistent, but generally curable, is observed in the course of pregnancy, or during or after delivery, and may or may not be accompanied by *eclampsia;* the lesion of the kidneys consists of a granular fatty degeneration of the epithelium comparable to that observed in scarlatina."

In a letter to the writer, dated May 5th, 1878, Prof. T. Gaillard Thomas, of New York, says, "During a practice of twenty-seven years in a large city, and connected with large hospitals during the whole period, I have of course seen a great many cases of puerperal convulsions. On two occasions only has albumen been absent from the urine in all these cases. In one it appeared two or three days after the convulsive seizure, in the other it never appeared.

"My belief is that puerperal convulsions are very generally due to poison

of the blood by elements of the urine, but that in exceptional cases any central or puerperal irritation which would create similar seizures in a child will produce them in the pregnant or parturient woman."

Dr. Ellwood Wilson, of Philadelphia, under date April 1st, 1878, writes, "I have seen no case of puerperal convulsions without albumen in the urine, although on some occasions when I had inquired of the attending physician whether there was albumen in the urine, and have been informed there was none, upon passing the catheter and testing the urine it was found to be heavily loaded with albumen."

Prof. Theophilus Parvin, of Indianapolis, writes April 18th, 1878, "In every case in which I examined the urine, there was found albumen."

Dr. Edw. L. Duer, of this city, has kept record of but five cases out of six which came under his observation. "In all of these cases there was present more or less albumen."

II. *Authors whose observations tend to controvert the view that puerperal eclampsia is due to Bright's disease.*

The theory that puerperal convulsions are reflex actions excited by cerebro-spinal or medullary irritation, of uterine origin, and transmitted through the ganglionic cells in which the reflex nerves terminate, is traced back by Dr. Thomas Moore Madden, in a paper read before the Obstetrical Society of Dublin, May 9th, 1874, to Laurence Joubert (*De Convulsionis Essentiâ et Causis*, op. An., p. 219, Ed. Antwerp, 1500), who was Professor of Medicine in the University of Montpellier in the middle of the sixteenth century. He asserted that the cause of convulsions is irritation, and that only by the removal of the cause of this irritation can the paroxysm be arrested.

The older English and American obstetricians generally claimed that puerperal convulsions were due to a determination of blood to the head, to be treated by blood-letting. This was the view of Scott (*Lectures on Midwifery*, 1775), Smellie (*A Collection of Cases and Observations in Midwifery*, 1779), Denman (*Introduction to Midwifery*, 1781), Foster (*Principles of Midwifery*, 1781), Bland (*Human and Comparative Parturition*, 1794), the Hamiltons (*Treatise on Midwifery, Hamilton's Lectures on Midwifery in the University of Edinburgh*, 1815–16. Reported by Dr. McKeever, MSS.), William Hunter Ryan (*A Manual of Midwifery*, 1831), Blundell (*Principles and Practice of Obstetric Medicine*, 1831), Davis (*Principles and Practice of Obstetric Medicine*, 1836), Burns (*Principles of Midwifery*, 1843), Maunsell (*Dublin Practice of Midwifery*, 6th Edition. Edited by M. Madden, M.D., 1871, p. 194).

Dr. Inglis (*Facts and Cases in Obstetric Medicine*, 1836) and others claimed that the circumstance that eclampsia commonly begins at night is a proof that the disease is connected with congestion of the brain.*

W. P. Dewees, M.D., in his *Essays Connected with Midwifery*, Philadelphia, 1823, and *System of Midwifery*, 1833, divides, as do several other older authors, puerperal convulsions into three different kinds: the epileptic, apoplectic, and hysterical species, according to the character which the convulsions bear. In his experience the "robust and plethoric women, and chiefly those of the lower class," are more liable to this disease, whilst "delicate and relaxed" women are seized generally with the hysterical species of puerperal convulsions, which occur less frequently and are less dangerous than the other species.

Dewees did not attempt to explain the cause of eclampsia, nor did he trace it to any causative lesion or to any changes in the blood.

He does not admit that pressure of the gravid uterus upon the descending blood-vessels, "causing a regurgitation of blood to the head, could produce convulsions, as this pressure is uniform in every pregnancy, and convulsions are comparatively of rare occurrence." He thinks, also, that pregnant women may be seized with convulsions from other causes than gestation.

Dr. Tyler Smith in his lecture on the cause of puerperal convulsions (*Parturition, and the Principles and Practice of Obstetrics*, 1849, p. 282), the substance of which was published in the London *Lancet* in 1844–45, before giving his own views presents those of cotemporary writers in England.

Ramsbotham (*Principles of Obstetric Medicine and Surgery*, 1842) says, "The most usual *proximate* cause of puerperal convulsion is probably *pressure on the brain*, this pressure being sometimes produced by the rupture of a vessel causing a sudden effusion of blood; sometimes by serous exudation into the ventricles or between the membranes; sometimes, and by far the most frequently, by *simple congestion of the cerebral vessels themselves*. But the disease has often proved fatal without any organic lesions being evident on dissection, and without even the vessels being observed to be preternaturally full. Into the *remote* causes it is not my wish to enter at any length, *because the subject is at best unsatisfactory, and little understood*. They have been ascribed to articles of food remaining undigested on the stomach, or irritation existing in some other

* For the facts in this paragraph I am indebted to the paper of Dr. Madden referred to, which is published in vol. ii. of *The Obstetrical Journal of Great Britain and Ireland*, April, 1874, to March, 1875, p. 236.

part of the alimentary tube; to general irritability of constitution; to a delicate and luxurious mode of living; to the depressing passions; to an overloaded state of the system; to over-distention of the uterus; to distention of the bladder; and to the death of the child. But the affection, in my opinion, originates most frequently in some deranged state of the uterus itself, probably in its nervous system, and consists in some irritation propagated from that organ to the brain."

Dr. Rigby (*System of Midwifery*, 1849). "The exciting cause of *eclampsia parturientium* is the irritation arising from the presence of the child in the uterus, or passages, or from a state of irritation thus produced continuing to exist after labor. The predisposing causes are general plethora; *the pressure of the gravid uterus upon the abdominal aorta;* the contraction of that organ during labor, by which a large quantity of the blood circulating in its spongy parietes is driven to the rest of the system, constipation, deranged bowels, retention of urine, previous injuries of the head, or cerebral disease, and much mental excitement.

"Also, in persons of hereditary predisposition, spare habits, irritable temperament, high mental refinement, and in whom the excitability of the nervous and subsequently the sanguiniferous system is called forth by causes apparently trivial."

Burns (*Principles of Midwifery*, 1837). "Convulsions of the kind I am considering evidently are connected with gestation or parturition; they occur at no other time, and are more frequent in a first labor. . . . They arise particularly from uterine irritation, but also seem frequently to be connected with a neglected state of the bowels. . . .

"A sympathetic irritation is almost invariably accompanied by an affection of the vascular system, productive of *great determination to the head*, either directly or indirectly, through the medium of the spinal nerves, which aggravates the evil, and becomes, indeed, the chief source of danger. I am inclined to think that, in a majority of instances, *the spinal cord is affected* by the state of the uterine nerves, and immediately afterward the head suffers. . . .

"A strong predisposition is given to this condition of the nervous system by a bad state of the bowels; and labor seems to bring the matter to a serious crisis. . . . On inspection after death, we sometimes find turgescence of the vessels of the brain or slight effusion of serum, but very often no mark of disease is to be discovered anywhere."

Dr. Lee (*Lectures on Midwifery*, 1844). "Those women are most predisposed to the disease who had had hysteria or epilepsy in early life, who have suffered from injuries of the head, or who have had violent attacks

of fever, with severe affections of the brain. Depressing passions of the mind appear to produce a predisposition to the disease. Unmarried women, who are excluded from society, and often addicted to the improper use of stimulants, are peculiarly liable to puerperal convulsions and mania. Terror and other violent mental impressions, and sometimes the pains of labor alone, are sufficient to excite convulsions. The disease occurs, not only in strong plethoric young women with their first children, in such as are of a coarse thick make, with short thick neck, but in weak, irritable, nervous females. There are some cases where irregularities of diet, especially the use of very indigestible food and stimulants, appear, without any other cause that can be discovered, to give rise to the disease. There are many cases in which the peculiar condition of the nervous system of the uterus appears to be the sole cause, and in all cases it is the principal predisposing cause, for the fits of convulsions occur in women in the first pregnancy and labor, and at no other time but during pregnancy and labor; and they often suddenly cease when the labor is completed, after every remedy has been employed without avail, except artificial delivery. *The condition of the brain on which the loss of consciousness and convulsions depend* is obviously produced by sympathy with the nervous system of the uterus; and the fits return and increase in violence till the uterus is emptied of its contents, and it is on them the irritation of the nerves of the uterus alone depends."

Dr. Fleetwood (*Theory and Practice of Midwifery*). "*It is exceedingly difficult to state anything very definite as to the cause of epileptic puerperal convulsions.* Doubtless they arise from the *sympathy of the brain* with the irritation of some different and often distant organ, it may be the stomach, the uterus, or the bowels. Intemperance in eating or drinking may give rise to it. Persons previously afflicted with convulsive affections are certainly predisposed to them at this time. Mental emotions and frights occasionally cause convulsions. In some cases, doubtless, they are owing to the efforts made during the labor-pains, by which an accumulation of blood takes place in the head. Atmospheric influence appears to have some effect in determining the frequency of the disease."

Dr. Locock (*Cyclopædia of Practical Medicine*, 1845). "The immediate causes of puerperal convulsions are often *very obscure.* They appear sometimes *to depend on a loaded state of the vessels of the brain;* at other times the brain appears to be influenced by distant irritation, either in the uterus or in the digestive organs; and again, in some cases, puerperal convulsions are induced by a peculiar irritability of the nervous system. The immediate attack may be brought on by a loaded or dis-

ordered stomach, or by food, however small in quantity, of an indigestible kind.

"Some substances, shell-fish for instance, have been found very frequently to induce convulsions in the puerperal condition, when at other times they may have been taken by the same individual with perfect impunity. A sudden fright, afflicting intelligence, or any unexpected or depressing mental emotion may excite the paroxysm. The violent straining caused by labor-pains, and even the disturbance of the frame by the earlier uterine contractions, *causing a temporary rush of blood to the head*, will sometimes bring on convulsions."

Dr. Collins (*Practical Treatise on Midwifery*, 1838), speaking of the relative frequency of convulsions in head presentations, observes, "This fact might be brought forward to support the opinion that puerperal convulsions were caused by the irritation produced in the dilatation of the mouth of the womb. This, however, is not the case, as we not unfrequently find patients attacked when the os uteri is completely dilated, and all the soft parts are relaxed. *I conceive we are quite ignorant as yet of what the cause may be, nor could I ever find on dissection any appearance to enable me to even hazard an opinion on the subject.*"

Dr. Merriman (*Synopsis of Difficult Parturition*, 1816). "There have been three especial causes assigned as usually producing this disease: 1st, General irritability of the constitution; 2d, Irritability of the uterus from distention; 3d, An overloaded state of the system. And practitioners have been influenced in their treatment of the complaint by the opinions they have entertained of its cause: thus, those who have attributed the convulsions to general irritability have considered opium as the proper remedy; those who have thought distention of the uterus the cause have recommended immediate delivery; those who believe an overloaded state of the system to be the cause of the convulsions employ large bleedings and other evacuants."

The views of Dr. Marshall Hall (*Constitutional Diseases of Females*, 1830) are also presented by the same author (Tyler Smith) as the basis of his own. The former writer says, "The principal causes of puerperal convulsion, besides the peculiar condition of the uterus itself, are indigested food, a loaded and morbid state of the bowels, a distended condition of the bladder, etc., mental shock or anxiety, muscular effort, hemorrhage," etc. Dr. Hall further believes that all these and similar causes act upon the *spinal marrow* and its system of excitor and motor nerves; and this view Dr. Tyler Smith proceeds to develop at greater length than Dr. Hall, but in accordance with his physiological and pathological doctrines. He concludes that

the convulsions are due to irritation of, or pressure upon, not the cerebrum, but on the medulla oblongata or the spinal cord; that this irritation may be either direct, or centric, or reflex. Among the centric causes operating directly on one or both of these centres he includes a toxic condition of the blood, produced by many causes, as constipation, asphyxia, etc. And among these latter he alludes to "Albuminuria, sometimes present, and which also appears to be caused by pressure on the kidneys and the renal vessels and nerves," and "accumulates noxious elements in the blood." No other allusion to the renal condition is made.

Ch. D. Meigs, M.D. (*Females and their Diseases*, Philadelphia, 1848,—"Obstetrics," 1852), expresses himself decidedly against the Bright's disease and uræmic theories. He thinks that pressure of the gravid uterus upon the greater abdominal vessels produces an "excess of determination of blood to the brain," which may cause œdema and convulsions. He rarely allowed his patients to lie on the back to be confined, so as to prevent an excess of pressure. Noticeable is the following sentence: "The profound insensibility (in eclampsia) ought to be regarded as an anæsthesia caused by the presence of much black blood in the brain; when it grows blacker, so as to render the patient dark like an Ethiop, the convulsion is nearer to its close; when circulating in the cerebellum the convulsions cease, and if it pervades the medulla oblongata the patient dies from want of power in the source of the vagus nerve."

M. Bach, in an unpublished memoir written about 1850 (?), thinks that puerperal albuminuria is only *sometimes* due to albuminous nephritis.

M. Blot (*Thèse de la Faculté de Médecine de Paris*, 1849) regards albuminuria as generally unconnected with Bright's disease. He also states that he has usually met with no disease of the kidney, and insists that in the majority of cases Bright's disease has no connection with eclampsia. Of 205 pregnant women whose urine he indiscriminately examined, he found albumen in 41 only.

M. Depaul (*Leçons de Clinique Obstétricale*, pp. 293–4) also reports cases of eclampsia in which albuminuria was absent, and takes much the same ground as Blot.

Dr. Breslau (*Monatsschrift f. Geburtsheilkunde*, 1860, p. 414) reports a case of eclampsia which occurred five days after labor. After the attack albumen was found in the urine, but *not before*. After the attack ceased, the albumen disappeared. The convulsions set in with so-called uræmic symptoms. The author states that the facts are adverse to the theory of Frerichs and Braun, for no unusual quantity of urea was found in the blood examined during the attack, and no carbonate of ammonia, while

the quick cessation of the albuminuria was against the presence of heart-disease. The albuminuria, he believes, was caused by the distended condition of the lower vena cava produced by the convulsions.

Dr. Ramsbotham (*Medical Times and Gazette*, vol. i., 1863) says that of 111 cases of eclampsia 79 were primiparæ. The urine was albuminous in every five cases out of six. The author makes the following remark: "We know that fibrine is in excess in the blood of pregnant women, we have reason to believe that albumen is also in excess; and from the observations of M. Blot, as a part of that excess is got rid of by the kidneys, we may easily imagine that if there be a much greater quantity formed than healthy blood ought to contain, the superfluous albumen may be the existing cause of the convulsive seizure." He reports cases of eclampsia where no trace of albumen was present.

The late Prof. Hugh L. Hodge (*The Principles and Practice of Obstetrics*, 1864, page 99) says, "So much importance has been attached to toxicæmia during gestation as a cause of eclampsia and other nervous affections that some additional facts, illustrative of this question, must be stated; and first, therefore, let it be remembered that all pathologists acknowledge that spasms, loss of consciousness, convulsions, etc., can be produced, in the most healthy individuals, by powerful impressions on the nervous system. Such disturbances are more common in women than in men, and occur more frequently in the pregnant than in the unimpregnated condition."

Dr. Hodge argues further that this disturbance may arise just as well from a "rush of blood to the head as from a deficiency of blood, as occurs in cases of anæmia, chlorosis, exhaustion, etc. Convulsions in a large proportion of cases arise from a congestion of the blood-vessels of the brain, or from an actual effusion of serum or blood into its substance or cavities."

Dr. Hodge does not think the toxicæmic theory of eclampsia is proved. He says, "There is no essential connection between albuminuria, blood-poisoning, and convulsions."

Dr. Hodge expresses himself also against the Bright's disease theory, as Bright's disease does not exist in a great number of eclamptic patients, and where nephritis does exist, convulsions are by no means the necessary consequence. He thinks that "the presence of albumen in the urine is no positive indication of nephritis or toxicæmia in the pregnant woman. This circumstance is merely the result of renal congestion or of general plethora."

The natural exhilaration of the nervous and vascular systems and the disposition to plethora constitute, according to Dr. Hodge, the predisposing

causes of puerperal convulsions. This predisposition, he says, is therefore created by, and dependent upon, the presence of a living child in the uterus, "the action of gestation." The predisposition ceases with the death or delivery of the fœtus, gradually diminishing, and, after a few weeks, disappears. Although strictly a nervous affection, yet there is great disturbance in the circulatory system, giving origin to congestion of the brain, spinal marrow, and other organs, which will greatly aggravate the original affection, even to fatal results.

Traube (*Berliner Klinische Wochenschrift*, 1864?) does not ascribe the so-called uræmic convulsions, even in renal disease, to the retention of excrementitious matters in the blood, but believes that the loss of albumen and the consequent hydræmia cause, through the simultaneous hypertrophy of the left ventricle, a greater pressure on the arterial system, which leads to *œdema of the brain*, which shows itself as coma when the cerebrum is œdematous, and as convulsions when the middle portions are affected. The convulsions are therefore due, not to disease of the kidneys, but to a deficiency of albumen in the blood and an increased pressure on the arterial system. And this is the cause of the convulsions of Bright's disease, according to Traube.

Rosenstein (*Pathologie und Therapie der Nierenkrankheiten*, 1te aufl., Berlin, 1843, 2te aufl., 1870) avails himself of this theory of Traube's to explain the phenomena of eclampsia.

"Very much like the influence of a valvular disease of the heart upon the kidneys," he says, "acts pregnancy. By the pressure which the pregnant uterus in the last month of gestation exerts upon the vessels of the abdominal organs, especially on the renal veins, the return of the venous blood is retarded, and thereby a stagnation in the kidneys as well as in the other organs caused."

Rosenstein agrees with Virchow, who (in his *Gesammelte Abhandlungen der puerperale Zustand das Weib und die Zelle*, p. 778) makes the following statement: "Certain it is that altogether too much attention is paid to the kidneys specially, and in the liver, at least, there can be quite as frequently noticed changes as in the kidneys. In both organs (the kidneys and the liver)—and, indeed, it is a question whether the spleen too ought not to be included with them—analogous parenchymatous swellings, due to the absorption of a granular, cloudy, and apparently albuminous substance into the interior of the glandular cells, are found, in consequence of which the whole organ becomes enlarged and diminished in consistency, and after its capsule is removed appears flabby. Very often these changes present an inflammatory character, and they may then be described as

parenchymatous nephritis or hepatitis. In other cases the inflammatory nature of the change is less apparent, and we must then be content to speak of them as an albuminous infiltration. In either case the secretory function of the organs suffers, and further research is required to decide which of the two exerts the greater influence."

Regnault and Devilliers (*Arch. Gén.*, 1848), already alluded to, also held the view that the liver too undergoes changes alongside with the kidneys (calling it nutmeg-liver).

Rosenstein thinks that there must be distinguished two kinds of kidney-affections in eclampsia: 1, Such which pre-exist eclampsia, where women who suffer from Bright's disease get pregnant, pregnancy thus being a complication; and, 2, Such kidney-affections as show themselves in perfectly healthy women during pregnancy or delivery (Bright's disease produced by pregnancy). The anatomical changes of the latter are those of a stagnation hyperæmia, and have nothing to do with inflammatory processes; in cases of recovery the secretory disturbances all disappear soon after the uterus is emptied.

The *cause of eclampsia* Rosenstein attributes to some *changes in the condition of the circulation of the brain* (not to blood-poisoning), and regards co-existing kidney-disease as a favoring but *not* as a causal nor as a necessary element.

He bases his view upon the following data: 1, The uræmic phenomena are not proven to be dependent upon the retention and the decomposition of the elements of the urine; 2, There are a considerable number of cases of eclampsia with healthy kidneys and without albuminuria, or the uroscopic signs of albuminuria did not precede but followed the attack (observations by Dorn, Winkel, Brammerstädt; 3, Pregnant women having Bright's disease sometimes pass through the delivery without eclampsia; 4, The peculiar circumstances and conditions in general under which eclampsia and albuminuria occur.

According to his theory, an increased pressure such as occurs during delivery, suddenly produced in the aortic system in cases where the blood is in a high degree of hydræmia, leads to hyperæmia of the brain. But, on account of the watery condition of the blood, œdema is the necessary result of the hyperæmia. The presence of serum in the tissues, again, exerts a mechanical pressure upon the blood-vessels and produces anæmia of the brain; *and the effect of this acute cerebral anæmia is a convulsion.*

Dr. Cairns (*Edinb. Med. Journal*, Feb. 1866) reports a case where convulsions were due to shock and hemorrhage (the placenta being ruptured), and *no albumen was found in the urine.*

Dr. Putégnat (*Journal de Méd. d. Bruxelles*, 1866) reports several cases of eclampsia where *no albumen* was found in the urine. He ascribes some of the cases to mental emotion.

Dr. Sourronille (*Arch. de Tocologie*, 1875, p. 498) mentions also a case of severe eclampsia in which no trace of albumen could be detected, and no œdema.

Dr. J. Braxton Hicks, in the *Transactions of the Obstetrical Society of London* for 1867, reports four cases of puerperal convulsions on which he founds a paper on the *Pathology of Puerperal Eclampsia.* In Case 1 the convulsions came on in the seventh month; delivery took place; the attack abated, and then ceased; about the sixth day, pneumonic symptoms appeared, and she died on the tenth day. The urine before the attack was free from albumen; twelve hours after, it was highly charged with albumen, with waxy and epithelial casts. Next day the urine was diminished, was higher colored, contained albumen, blood-disks, and uric acid crystals. This continued for six or eight days, the albumen gradually disappearing before she died. There was no anasarca before the first attack, but she had some unpleasant sensations in the head for a day or two before, with partial loss of sight, etc.

Case 2 was attacked in the early part of her first labor with puerperal eclampsia repeated with great frequency, and delivery was ultimately accomplished by craniotomy; the attacks gradually subsided and the next day she became sensible. The urine drawn during the early part of the case contained not a trace of albumen; that drawn on the first and subsequent occasions after delivery contained large quantities of albumen. After cessation of lochia there was no albumen. No microscopic examination. No œdema or other symptoms beforehand.

Case 3 during application of instruments had an epileptiform fit, twice repeated after labor. Urine, after first attack, had so small a trace of albumen that it was doubtful. Nine hours after, its presence was strongly marked. Twenty-eight hours after the first attack no trace of albumen was detectable. Recovery rapid. No abnormal symptom before confinement.

Case 4, a girl of 14 years at full term, had convulsions of great severity, when the head was near the outlet. The urine drawn after the placenta was expelled contained no albumen. The fits continued, with coma between. The urine drawn off twelve hours after the first time contained copiously of albumen.

There was no *post-mortem* examination of either case which died. Yet in the paper alluded to he says, "But that in a certain proportion of

cases neither chronic Bright's disease nor any organic affection of the kidney has existed, the cases I bring forward will prove."

Quoting in connection with these cases some experiments, according to which twenty-four hours must elapse after the kidneys have ceased to act before symptoms of uræmic poisoning can occur, he says, "If this point be granted,—and it seems that, so far as our present knowledge goes, it must be,—then the only modes of explaining the occurrence of acute nephritis are in one of these three ways: either, 1st, that the convulsions themselves are the cause of the nephritis; 2d, that the convulsions and the nephritis are produced by the same cause,—*e.g.*, some detrimental ingredient circulating in the blood, irritating both cerebro-spinal system and other organs at same time; 3d, that the highly-congested state of the venous system as is produced by the spasm of the glottis in eclampsia is able to produce the kidney complication."

Winkel (*The Pathology and Treatment of Child-bed*, Rostock, 1869. Translation by Chadwick, 1876) takes a very decided view opposed to the uræmia theory. He says, "The *condition of the brain*, as revealed at the autopsies of those dying of eclampsia, is, in the first place, that of extreme anæmia with more or less marked œdema and obliteration of the convolutions; much more rarely (in only about one-sixth of the cases) there has been found extensive hyperæmia, or even capillary ecchymoses and apoplectic deposits as large as pigeons' eggs. The brain has very rarely (2–3 per cent.) appeared to be perfectly healthy. The condition of the kidneys is, on the other hand, almost the opposite, since in 35.7 per cent. these organs have been found healthy, and only in 64.4 per cent. were there detected any decided lesions (Brammerstädt). The comparative number of sound kidneys is, therefore, considerable, being more than one-third."

The urine contains as a rule (in 84 per cent.) albumen; but albumen is found sometimes also in normal and perfectly healthy pregnant women. Albumen appears often only after the attacks are over. *Thus the presence* of albumen in the urine is by no means a constant phenomenon of eclampsia; nor is there any greater uniformity in the presence of casts. Winkel agrees with Traube and Rosenstein that in most cases eclampsia is due to an affection of the brain.

Niemeyer (first American, 1870, from seventh German edition, and probably previous editions) says, "We are quite ready to admit that in most cases of *eclampsia puerperarum* there is renal disease; but we deem it quite inadmissible to attribute the albuminuria, dropsy, and eclampsia to Bright's disease."

Schroeder (*Manual of Midwifery*, English edition, 1873, from the third German) is best satisfied with the theory of Traube and Rosenstein, already given, but says also that the etiology of these convulsions is by no means sufficiently known, and that it is highly probable that the clinical picture here called eclampsia may in future be broken up into several morbid processes widely differing in an anatomical and pathological point of view.

Dr. S. Cummins (*Arch. de Tocologie*, 1874, p. 694) describes a case where labor-pains produced *general* emphysema (emphysema of lungs proper being a constant sequel of eclampsia), and argues that the emphysema causes a venous congestion all over the body and in the kidneys, too, where the efferent veins of the Malpighian bodies cannot empty themselves freely into the renal veins, and produce albuminuria, but the latter is not the cause but the *consequence of the convulsions*. He found eclampsia to occur most often in illegitimately pregnant women.

Dr. Martell (*Arch. de Tocologie*, 1876, p. 184) found only a trace of albumen in a case of eclampsia which terminated in death. On *post-mortem* examination he did not find any changes in the kidneys.

In another case reported by him (the same *Arch.*, 1877, p. 236) he found the kidneys a little congested, and slight discoloration of the cortical substance.

The late Prof. Joseph Carson, in a review on puerperal eclampsia (*Amer. Jour. Med. Sciences*, April, 1871) concludes from a careful study of the subject that puerperal eclampsia and epilepsy are analogous affections, and that neither the determination of blood to the head nor urea or carbonate of ammonia constitute the cause of convulsions. He does not admit any connection of puerperal eclampsia to kidney-disease, and considers albuminuria—which he regards as identical with hemorrhage—only as far as its relation to anæmia. Anæmia of the brain he considers to be the causal lesion of eclampsia.

Dr. Hiram Corson, of Conshohocken, Pennsylvania, who has seen very many cases of puerperal eclampsia in a large country practice extending over nearly fifty years, and who has published the cases he had prior to the year 1857 in the *Transactions of the Pennsylvania State Medical Society*, under the title *Midwifery in the Country*, writes, under date of April 3d, 1878, "I have almost no belief in albuminuria preceding convulsions. I would in every case of a pregnant woman who had the symptoms of deranged or diseased kidney, and who, from that cause, seemed threatened with convulsions, treat her, not for poison in the blood, but to relieve a deranged nervous system. The vice is in that, not in the kidney, which is only secondarily affected."

Prof. Ellerslie Wallace, of this city, in a communication to the writer dated April 23d, says, "I have seen several cases of albuminuria in pregnancy where there were dropsy, etc., and in some cases strongly expressed. Treatment, during the latter weeks of pregnancy, relieved, but did not remove the albuminuria. I was anxious about such cases, of course, and I placed such women under anæsthetics in the early part of the second stage of their labors, and delivered them by the forceps. And in not one of these cases did I see puerperal convulsions.

"I have seen puerperal convulsions plainly induced by the excessive pain of the labor, in several instances, in women who had passed through their pregnancies without any unpleasant symptoms, and in whom there *might* have been, more than ordinarily, albuminuria. But they showed, during pregnancy, no symptoms to demand investigation as to the renal function. I saw, several years ago, a few cases of puerperal convulsions from the use of ergot in first labors, where the uterine action under ergotic excitement was severe and painful."

Prof. R. A. F. Penrose, of this city, writes, April 24th, 1878, "When a woman becomes pregnant, suffering, at the time of conception, from any of the several disorganizing conditions of renal disease, grouped roughly in the generic term of 'Bright's disease,' the developments of gestation necessarily greatly aggravate the pre-existing kidney trouble, and uræmic convulsions are not only possible, but probable, the liability to them increasing as gestation advances.

"There is *not*, however, *any necessary* or *definite* relationship between so-called 'Bright's disease' and puerperal convulsions. Individuals differ so greatly in their nervous peculiarities, as well as in the effects which pregnancy occasions, that the same condition constantly produces very different results in different constitutions.

"Albuminuria, nevertheless, whether due to renal degeneration (Bright's disease), or whether the result of that hyperæmia caused by pressure, or sympathy, or by both, should in all pregnant women excite solicitude, since it indicates imperfect performance of essential renal functions, and a consequent failure in elimination that may in some constitutions lead to convulsions, and in others may develop those innumerable nervous symptoms, of greater or less gravity, which we meet with so frequently.

"While holding these views as to the dangers attending a failure in the proper performance of the functions of the kidneys,—whether this failure be owing to chronic and incurable conditions (Bright's disease), or to temporary and remediable ones,—I also *know* that there are many cases of puerperal convulsions brought on by causes entirely independent of all

kidney disturbance. The explanation of such cases is to be found in the fact, to which I have referred, relative to the great diversity of effects caused by gestation in different constitutions. Powerful emotions, alone, have in my own experience caused terrible and fatal cases of puerperal convulsions.

"To give my individual experience: I will say that in most cases of puerperal convulsions which I have seen the kidneys were in fault,—that is, there was albuminuria; but not always, or even not often, Bright's disease. In some of my cases no albumen whatever was found in the urine. A very interesting case of this kind occurred in the wards of Blockley Hospital. The urine of this woman had been examined almost daily, for some weeks before her labor, by Dr. D. F. Woods, who was conducting an extensive series of observations on the urine of gestation at the time, and was examining, almost daily, the urine of all the pregnant women in the ward (forty-six in number). This woman's urine before her labor presented an average sp. gr. of 1024; was acid; *at no time* contained albumen, but did contain uric acid and urates in excess at labor. She had as dreadful convulsions as I have ever witnessed; was comatose for several days after. Ultimately she recovered completely."

Prof. W. T. Lusk, of New York City, writes, April 20th, 1878, "I have had cases of puerperal convulsions in which careful examination beforehand proved the absence of albuminuria. I could not say in what proportion of cases such absence exists. I have met no case in which albuminuria did not make its appearance after the outbreak of convulsions."

III. *Writers who admit that puerperal eclampsia is sometimes due to Bright's disease, but who believe that it occurs as often independently of such cause.*

Scanzoni early took part in the discussion of this question, and at first (*In der Klinisch. Vortrag. über special. Path. und Ther. d. K. des weibl. Geschlechtes*, Prague, 1854) took decided ground against the view that Bright's disease is an essential lesion in most cases of puerperal eclampsia; but more recently, in the fourth edition of his *Lehrbuch der Geburtshülfe*, Wien, 1867, says, that for more than ten years he has advocated uræmic (not carbonate of ammonia) intoxication as the cause of eclampsia; although, sometimes, nervous reflex excitability plays a very important *rôle* in the causation of eclampsia.

The uroscopic phenomena are indeed seldom wanting; albumen and tube-casts are usually found, and always in direct proportion to intensity and duration of the attacks. The whole complex of symptoms in eclampsia

is such that we may conclude upon a derangement of the urine-secreting function of the kidney.

Pathologo-anatomical observations show that perfectly healthy kidneys are in this disease very rare, although very marked lesions are also infrequent, and seldom only do we meet with the third stage of Bright's disease (atrophy).

The second stage (stage of exudation), even, is seldom well pronounced, and unequally distributed through the parenchyma of the organ. Often no other tissue-changes are found than a simple hyperæmia and swelling of the kidneys. The anatomical changes, indeed, often leave us in doubt whether they had been intense enough to cause the uræmic phenomena. But it is said that the process in the kidneys being a very rapid and acute one, the severest functional disturbances result as suddenly, which is not the case in a chronic kidney-disease, where the condition is gradually developed.

"This explanation," says Scanzoni, "is very plausible; and in favor of it are also the phenomena of the acute attacks of Bright's disease following cholera, typhus, scarlatina, and which not unfrequently terminate in death by so-called uræmic phenomena, whilst at the *post-mortem* examination the kidneys do not show any other changes than those of the first or the beginning of the second stage of Bright's disease."

Another circumstance to which Scanzoni directs the attention, and which, as he says, has been heretofore overlooked, is the abnormally-elevated reflex irritability which is peculiar to the pregnant woman, and which in the presence of even a slight functional disturbance of the kidneys, and the consequent anomalies in the composition of the blood, exert such a harmful influence upon the central nervous system as under other circumstances could occur only in very far advanced parenchymatous changes of the kidneys.

The cause of the elevated reflex irritability in the pregnant woman Scanzoni considers to be *the general disturbance in the nutrition* due to abnormal condition of the blood.

The blood of pregnant women is characterized, as it is known, by an increase of water, of fibrine, and of white blood-corpuscles, and by a diminution of red blood-corpuscles and of albumen, or, in other words, it shows the peculiarity of the blood of anæmic women; and it is a known fact that nothing favors more nervous derangements and all kinds of convulsions than anæmia.

The brain of eclamptic women is found, in cases where the convulsions did not produce marked circulatory and respiratory disturbances, as a rule to be very dense, anæmic, and more or less infiltrated with serum.

Doubtless there is some connection between the imperfectly-understood disturbances of nutrition of brain-substance, and the primary disease of the parenchyma of the kidneys.

"According to the present state of our knowledge of eclampsia," further says Scanzoni, "we come nearest to the truth if we define the cause as follows: eclamptic convulsions are in the majority of cases produced by the accumulation of excrementitious matters produced by disturbed function of the kidneys; but whether here the urea, the known or some still unknown extractive matters, or the carbonate of ammonia plays an important *rôle*, must be left to future researches.

"But what explanation," he asks, "shall be given to those cases of eclampsia, becoming in recent time more and more frequent, in which clinical observation and anatomical examination exclude all possibility of a kidney-disease and uræmic intoxication?"

Some of them are not true eclampsia, but those which are can be due to elevated reflex irritability of the peripheral nerves, and to irritation of the central nervous system and of the brain. Scanzoni thinks that these play an important *rôle*, not only in these exceptional cases, but in every case of eclampsia.

There are also some cases in which the cause of eclampsia had to be ascribed to hyperæmia of the brain and of the medulla oblongata, as besides this condition no other lesion (and no kidney-changes) could be detected. As far as the influence of the convulsions upon the state of the circulation in the kidneys is concerned, Scanzoni thinks that albuminuria may be also the consequence of the convulsions, and thus cautions not to regard albuminuria in eclamptic patients as indicating invariably Bright's disease, especially in the absence of tube-casts.

In connection with the etiology of eclampsia, Scanzoni thinks pressure to be an important causative element. He does not, however, refer to pressure upon the kidneys and the renal blood-vessels, as many other authors do, but to pressure and to tearing of nerves, chiefly of the uterine nerves during the contractions of labor, by which reflex convulsions may be very easily excited.

Thus it will be seen that although at first he very distinctly announces himself in favor of Bright's disease as the cause of puerperal eclampsia, he is still unwilling to attribute all cases to it.

Dr. Barnes (*Lectures on Puerperal Fevers. Lancet*, 1865) regards albuminuria as being evidence of the kidneys being overpowered by the double strain of having to serve as emunctories for two organisms, and thinks it probable that sometimes albuminuria and uræmia are of sudden

origin, and that the convulsions follow almost immediately upon a failure of the kidneys.

Later, however, Dr. Barnes (*British Med. Journal*, 1873) says, "Convulsions in pregnancy and labor may occur *without albuminuria*. Albuminuria may exist in pregnancy, and go on to the end without necessarily inducing either convulsion or labor.

"In some cases the albuminuria existed before the pregnancy, depending upon chronic Bright's disease. Of course, the pregnancy does not mend matters. The albuminuria persists. But if I may trust the few observations to the point which I have been able to make, convulsion is less likely to ensue than in the rapidly-produced acute form. We may imagine that in chronic Bright's disease a process of accommodation takes place, whilst in acute albuminuria the nervous centres suddenly invaded by poisoned blood are unprepared for resistance."

Chailly-Honoré (*Treatise on Obstetrics*, Paris, 1867, p. 173) states that he is inclined to adopt M. Blot's view on albuminuria in pregnancy (see page 192). He says also, however, that the urine in a majority of cases is very much like that in scarlatina, where a similar congestion of the kidneys is observed. This is the character of two-thirds of the cases. Although somewhat at a loss, therefore, in which category to place him, the writer has included him in the third.

Prof. T. A. Reamy (*Transactions of the Ohio State Medical Society*, 1868, abstract in *Half-Yearly Comp. of Med. Sci.*, 1869) says, "Puerperal eclampsia and Bright's disease of the kidneys rarely ever stand in the relation of cause and effect, unless we understand simple and temporary congestion of the cortical portion of the kidneys, resulting from mechanical pressure, to constitute Bright's disease.

"True Bright's disease is sometimes found affecting women; such cases generally proving fatal, with or without convulsions. The attack is usually precipitated by the nervous state consequent upon the nature of the blood and character of its circulation reaching a point where the elements of parturition are as the torch to a magazine. There are deranged secretion and excretion in a certain proportion of cases involving the product of hæmatosis."

Naegele and Greuser (*Treatise on Obstetrics*, Paris, 1869) state that the *autopsies* in cases of death from eclampsia in general do not give any explanation of the nature of the disease. As far as the kidneys are concerned, they are either one or both in a state of hyperæmia. Sometimes they present the first or the second degree of Bright's disease (apoplectic spots and beginning of exudation in the tubules or fatty infiltrations); the third degree—that is, atrophy of the kidneys—is rare.

Dr. S. Busey, in a paper published in 1870 (*Artificial Induction of Labor in Uræmia*), is decidedly in favor of the Bright's disease theory in eclampsia. He says, "It seems to be established:

"1. That albuminuria in pregnancy is either the immediate result of pressure of the gravid uterus, or that incipient renal degeneration or precursory tendency thereto is aggravated and determined by the co-existence of pregnancy.

"2. That certain changes take place in the constitution of the blood consequent upon pregnancy or consecutive therewith, which alterations increase in intensity during the progress of gestation, and that renal disorganization, as cause or effect, may and frequently does co-exist.

"3. Even though no renal lesion exist primarily, the long-continued operation of causes which induce albuminuria in pregnancy may and frequently does result in organic renal disease; so that, in reality, it is not so much a question of the identity of albuminuria in pregnancy and Bright's disease in their visible phenomena, as it is whether they are the effects of the same proximate causes."

Concerning the blood-change theories, Dr. Busey remarks, "It matters not whether it be a blood-disease, showing itself at first simply in the excretion of albumen through the kidneys, and eventuating in organic renal disease, or primarily a nephritis; if dependent upon utero-gestation, the latter is both the exciting and predisposing cause."

Dr. Busey reports eight cases of puerperal eclampsia, seven of which he considers undoubtedly as uræmic convulsions and illustrating the different stages of the renal lesion; but in one case there was not found any albumen, either before or after the attacks, and thus he considers that the convulsions were induced by some other exceptional cause.

In this paper Dr. Busey believes "that eclampsia is but the accessory phenomenon of Bright's disease, which is the first link of a chain of morbid changes leading to it."

In a more recent paper, however (*Address on Obstetrics*, etc., read before the American Medical Association, 1876), he has evidently changed his views, and admits that numerous causes other than those growing out of Bright's disease may produce convulsions. Although he here also states that ninety of the cases of puerperal eclampsia are associated with albuminuria, and that much the larger number of the *autopsies* of women dying of puerperal convulsions exhibit renal lesions.

It is not easy to arrive at Dr. Busey's precise views on this subject from this more recent pamphlet; but he says (p. 27), "The physiological phenomena which favor cerebral congestion lend force to the once very

commonly accepted theory that puerperal convulsions were occasioned by a determination of blood to the head. This view derives important corroboration from the anatomical resemblance of the arterial cerebral circulation in women and in the cow, to which parturient apoplexy and convulsions are mainly confined."

In a letter to the writer, dated April 17th, 1878, he writes, "I have been much interested in the precise question which you propose to discuss, and at one time held very advanced views, maintaining that puerperal albuminuria was a symptom of structural changes identical with those which occurred in Bright's disease. This opinion I have modified somewhat. But to determine the ultimate relation of the two affections I have been for several years collecting the *autopsies* of those who died from puerperal eclampsia, and as yet have not found one which does not exhibit renal disease."

Strongly as these latter remarks point to the views of the writers of the first category, it seems necessary, in consideration of the matter of the second pamphlet and what precedes them in his letter to the writer, to place Dr. Busey in the third category.

Prof. Fordyce Barker (*The Puerperal Diseases*, New York, 1874), in the chapter on puerperal albuminuria (p. 68), says, "Albuminuria often exists without causing convulsions; dangerous and fatal convulsions may occur when albuminuria is wholly absent; and, still further, the nervous perturbation which causes the convulsions may also be the cause of albuminuria, or even the convulsions themselves may be the cause of albuminuria. In the present state of science, albuminuria is no longer believed to be a symptom of Bright's disease only. In fact, it is probable that, in nineteen cases out of twenty of puerperal albuminuria, the structural lesions of the kidney implied in the term Bright's disease do not exist.

"Robin, in his recent work on the fluids of the body, has demonstrated that urinary albumen has not the same composition as the albumen of the blood, and that the albumen of Bright's disease differs essentially from that occurring in temporary albuminuria of pregnancy, as can easily be shown by its chemical reactions. The albumen of the urine in Bright's disease, when brought in contact with the oxide of copper, assumes a beautiful reddish-violet color, and produces a more or less abundant flocculent black precipitate. Now, the urinary albumen of pregnancy, when Bright's disease does not exist, while it coagulates readily by heat and nitric acid, does not exhibit any such reaction with the oxide of copper. So, also, Robin has demonstrated that granular casts are not characteristic of any particular morbid state or pathological change of structure of the kidneys."

Barker also found often the urine after the convulsive attacks during labor loaded with albumen, whilst there had been no traces of albumen during pregnancy. He believes also "that we meet with convulsions, developed by emotional causes, unassociated with any anatomical lesions."

Barker directs, also, the attention to Frankenhäuser's work *On the Nerves of the Uterus*, based on careful dissections and illustrated by most beautiful plates, in which is demonstrated a direct connection between the nerves of the uterus and the renal ganglia. Frankenhäuser reasons from his discovery that the theory of pressure on the abdominal and renal vessels is highly improbable; he thinks that albuminuria and eclampsia are due to the excitation of the uterine and renal nerve plexuses. Dr. Barker adds that this theory seems to have been anticipated many years before by Dr. Tyler Smith, of London, who suggested that the albuminuria "may depend upon sympathetic irritation of the kidneys by the gravid uterus, similar with the irritation of the salivary glands, the mammæ, the thyroid, etc., and not upon mere pressure."

"Clinical observations have established the fact that the following conditions are predisposing causes of eclampsia: viz., albuminuria, hydræmia, anæmia, uræmia, and primiparity. Perhaps I should add hereditary and atmospheric influences. The latter have been alleged as a predisposing cause also by Andral, Dugès, and other French authors, and by Smellie, Denmann, Ramsbotham, Davis, and Simpson."

Dr. Barker also reports a case of puerperal eclampsia which terminated fatally, although there was not even temporary albuminuria, nor other symptoms of Bright's disease. The kidneys were examined after death by Prof. Alonzo Clark, "who pronounced them slightly congested, but in other respects perfectly healthy."

Two other cases are reported by Dr. Barker, in both of which the symptoms of Bright's disease were present, and in one of which the diagnosis was confirmed by the autopsy. In the remaining case, which recovered, there were œdema, convulsions, and albuminuria, which persisted eleven days after delivery.

In a letter to the writer bearing date April 30th, 1878, Prof. Barker writes, "I have seen quite a number of cases in which no albumen has been discovered in the urine until after the convulsions had appeared, although I have been assured that the urine had been tested several times previous to the convulsions. In my hospital practice, in thirty-four cases of convulsions no albumen could be detected in the urine after repeated examination in eight. In four of those albumen appeared after the convulsions, lasting, however, but a few days. In my private practice, in

two cases of convulsions repeated examinations failed to detect either albumen or casts; but in three both albumen and casts appeared after the convulsions."

Dr. George J. Engelmann, of St. Louis, writes, April 20th, 1878, "I do not recall a single clear case of puerperal convulsions without albuminuria, but I do know that a patient with Bright's disease with albuminuria need not necessarily have convulsions in child-bed or during labor. Convulsions may occur during the stage of dilatation without albuminuria, due to rigidity or irritation of the os, which cease upon complete dilatation. These are reflex neuroses,—hystero-neuroses, as I call them.

"I have very carefully examined the kidneys in several cases in which death resulted, and the urine contained albumen. (I still have the specimens.) As far as I remember, the kidneys were somewhat congested, but showed no structural change."

Conclusions of the Writer.

It might be anticipated that I approach hesitatingly to conclude upon a subject on which the results of observation are so various, and on which so many better qualified have thought carefully and expressed themselves so diversely. The following I have, however, reached:

First.—There are no reasons why we should exclude from the causes of convulsions in the puerperal state those which operate to produce convulsions in the non-puerperal condition. This more particularly when we admit, as I think all must, that, let the cause be what it may, the nervous centres of the pregnant woman are generally hypersensitive, and therefore more ready to respond to peripheral stimulus—irritation of any kind—than are the nerve-centres of non-pregnant women; and this may be increased by the pressures and congestions incident to labor. This being admitted, any such peripheral irritation as the pressure of a child's head upon a rigid os, like the pressure of a tooth upon a child's gum, may excite a convulsion; or the irritation of uterine nerves compressed during muscular contraction, or emotion, whether pleasurable or painful, distress, anxiety,—all *may* excite a convulsion. And it is not impossible that such a convulsion may be fatal, as it is not impossible that such a convulsion may be fatal in the non-pregnant woman. But such a result is indeed rare, and convulsions from these causes are not generally serious. They occur most often in primiparæ, where the labor has been long and painful.

But may such convulsions as these occur after delivery, when the irritation has apparently subsided? I believe they may. For, in the first place, the irritation does not necessarily cease with the termination of the

labor. The sting of the lash by no means ceases with the omission of its strokes. And, in the second place, we know that some time is often required after the operation of a peripheral irritant through a nerve upon a nerve-centre before the latter responds. It would seem as though some time must elapse before the requisite impulse can be generated in the ganglion cells to produce an explosion the resultant of which is a convulsion. Such I believe to be the nature of the convulsive attacks in most cases reported, where there is no albuminuria before the attack, and but little after it.

Second.—I think it not impossible, even, that puerperal convulsions may be caused by the congestion to which these same centres are subjected in a hard labor, as is evidenced by the red face, the protruding eyeballs, and headache, often agonizing during a pain, which the muscular effort produces. The number of these—the congestive or apoplectic cases of the oldest authors—has, however, in the admission of all, been reduced to a minimum by the subtraction from them of the cases of reflex convulsions just referred to in my first category.

Third.—Outside of these categories, in which I would include a limited number of comparatively harmless cases and a smaller number of more serious ones, I would assign the causal lesion of puerperal eclampsia to be Bright's disease of the kidneys. The Bright's disease which I believe to underlie the large majority of serious cases of puerperal convulsions may either be present at the time the woman becomes pregnant, have preceded the pregnancy, or it may be acquired during the pregnancy. In the former case, as Dr. Barnes has said (*loc. citat.*), "the pregnancy does not mend matters," and the tendency, at least, is, by reason of the pressures and congestions naturally present in pregnancy, further to interfere with the elimination of excrementitious matters, whose secretion is already embarrassed by the renal lesion, independent of any addition thereto from the pregnant state. And yet it is a fact observed by very many that such persons by no means necessarily have convulsions before, during, or after labor, especially if they are multiparæ. This is unquestionably due to the accommodation or balance which we know to be set up in different parts of an economy where another becomes gradually involved in disease. We all know how different are the results of gradual and sudden brain-lesions, —how the former may advance to an extreme degree without giving symptomatic evidence of their existence, and how serious are the consequences of even slight degrees of the latter. But we must at least admit the pregnant woman with pre-existing Bright's disease to be in the same danger of convulsions as the woman with Bright's disease who is not

pregnant, and it is not unreasonable to suppose that the danger of the former is somewhat greater than that of the latter, and in cases of primiparæ very much greater. And thus are caused some cases of puerperal convulsions. In these cases the form of Bright's disease may be any one of those to which all are subject.

In the second series,—where the disease is *acquired*,—it is almost invariably catarrhal nephritis (parenchymatous nephritis, or tubal nephritis), of which the typical example is seen in the Bright's disease concurrent or sequel to scarlet fever.

This conclusion—that most cases of puerperal convulsions are caused by Bright's disease—is justified by the fact that these cases are almost invariably accompanied by *albuminuria*, and, where a microscopic examination of the urine is made, by *tube-casts;* that a very large number are also attended by œdema; and that where they terminate fatally the autopsy generally reveals disease of the kidneys. In confirmation of this, while referring generally to the cases reported in the past pages, I desire to call your attention particularly to an analysis of the series of cases, one of the largest on record, as well as one of the most accurate,—the microscopic and chemical examinations being often made by Prof. Flint, Jr., and that of the kidneys often by Prof. Alonzo Clark, of New York,—reported by the late Prof. Elliot, in his *Obstetric Clinic* (1868), pp. 101 to 126. The list includes fifty-one cases of albuminuria and eclampsia, of which six have no exact bearing upon the subject. Eleven are cases of kidney-disease, with albuminuria, during pregnancy and parturition, not associated with convulsions, thus leaving for consideration thirty-four cases of eclampsia. In four out of these thirty-four cases eclampsia was not associated with albuminuria, nor were any tube-casts in the urine. Of the thirty cases in which the albuminuria and eclampsia co-existed, fourteen recovered and sixteen died. *Autopsies* were made only in seven cases, with the following result: Case 3. One kidney in a state of advanced Bright's disease, and the other perfectly healthy. Case 5. Advanced Bright's disease (large white kidney). Case 11. Well-marked Bright's disease. Case 13. Fatty kidney. Case 34. Kidney enlarged and congested, but not changed in structure. Case 36. Kidney large, white, congested; weight of the two, thirteen ounces, and, under the microscope, granular degeneration. Case 47. Although albuminuria, œdema, no symptoms of uræmia; there was no convulsion until the next day after labor; coma supervened immediately after the convulsion, and she died the next day, forty-two hours after delivery. At the *autopsy*, "the kidneys weighed four and a half ounces, and were healthy under the microscope." Both lateral ventricles of the

brain were filled with bloody serum; the third ventricle contained serum and a small clot; and the fourth was filled with clotted blood. The vessels of the neighborhood were examined by Prof. A. Flint, Jr., and found to be atheromatous.

The notes on the four cases of eclampsia referred to above which were not associated with albuminuria, are as follows: Case 8. Has had three miscarriages; there was œdema of the face and upper extremities; secretion of urine normal in quantity, and no albumen present; no microscopic examination of the urine was made; eclampsia; child putrid; death of the mother; no *autopsy*. Case 19. Ninth confinement; no albumen, but great quantities of urate of ammonium, some urate of sodium, and bile; eclampsia; mother and child lived. Case 30. Primipara; no albuminuria; only two convulsions; recovery. Case 39. Primipara; eclampsia after delivery; only three convulsions; no loss of consciousness; no albumen; recovery.

Of the eleven albuminous pregnant women referred to in whom no convulsions occurred, only five recovered, and six died during or after delivery. *Autopsies* were made in three only, and in *each was found advanced renal lesion*.

Now, as to Elliot's Case 47, where there were albuminuria, œdema, a physiological confinement, a convulsion the next day, followed by coma which continued until death, and the *post-mortem* revealed the *kidney weighing four and a half ounces, but healthy under the microscope*, there were, however, brain-lesions, including a small clot on one of the ventricles, and the fourth ventricle filled with clotted blood. The vessels in the neighborhood were examined by Dr. A. Flint, Jr., and found *markedly atheromatous*. It is not stated whether the kidneys singly weighed four and a half ounces or jointly; most probably the former is intended; but in either event they would seem small. *They were not examined microscopically*. The vessels of the brain were atheromatous, and death occurred by rupture of one of them. Were these not, then, cirrhotic kidneys, attending which we constantly have atheroma of the vessels of the brain and death by apoplexy?

As to the remaining four cases in which there was no albuminuria, it will be observed that in one there was a putrid child, and no examination of the urine seems to have been made after the convulsions set in, and no microscopic examination at any time; no *autopsy*. There may still have been Bright's disease, or the convulsions may have been due to the absorption of septic matter from the putrid child. The remaining three cases were mild cases, and may be placed in my first category.

Again, take the three cases of Prof. Barker, alluded to under the abstract of his views. In two of the three, Prof. Barker acknowledges Bright's disease, and in one of these the diagnosis was confirmed by a *post-mortem* examination, in which the kidneys were found very much diseased. In the third case there was no albumen in the urine drawn from the bladder after she had had one fit, and again after she had had fifteen at least, recurring at intervals of five or ten minutes, and being profoundly comatose in the intervals. Premature labor was induced, but she died after further convulsions. At the *post-mortem* examination there were two ounces of serum in the cavity and lateral ventricles of the brain, and the cerebral vessels were congested, but the kidneys were pronounced by Dr. Alonzo Clark "slightly congested, but in other respects perfectly healthy." Here, then, is a case of severity, in which there is no albuminuria, and no renal lesions are discoverable after death. These are, at most, not very numerous. They may be slightly reduced by cases of the kind included in the first category, in which, without appreciable lesion, death occurs, just as it sometimes occurs by convulsions in non-puerperal women without any discoverable lesion; and it is not impossible that this one of Prof. Barker's, which I confess is the most striking I have met, may be of this number, especially as the case was one of peculiar distress,—that of a young woman of evident refinement and education, who had wandered from her home several hundred miles distant, and, friendless in a large city, was compelled to make her bed on a door-step, whence she was taken roughly to a police-station, in which she had her first fit. Certainly, if circumstances of this kind are ever sufficient to excite convulsions, these are such. But I believe the number may be still further reduced when we remember the imperfect character of the examinations both of the urine and the kidneys. Those only of us who have had experience in hospitals know how carelessly and hastily these examinations are often made. And in the examination of the kidneys themselves the liability to error is still greater. I hold that almost never is a naked-eye examination of a kidney sufficient to justify an assertion that it is not diseased, and especially when the naked-eye examination reveals "congestion." The microscope should always be used in these cases. For this very congestion obscures more delicate changes, so as to make them undistinguishable without the microscope, and the microscope used intelligently. And, further, can we assign a limit to the mischief of a simple congestion in a kidney, especially if that congestion be suddenly induced? Finally, if it is remembered that experience often shows temporary absence of albumen in some cases of chronic Bright's disease, and that cases have even been reported with albuminuria, dropsy,

uræmia, and death after scarlet fever, and yet the *autopsy* discovers no lesion in the kidneys,—when all these matters are considered, *I cannot but think that the number of serious cases of puerperal eclampsia which cannot be attributed to some form of Bright's disease is small.*

If I am asked whether I think it impossible that a convulsion should cause albuminuria, I answer, By no means. On the other hand, we not only have every anatomical reason for supposing this possible when we recall the increased vascular pressure which must result from a convulsion, but we have also clinical evidence to this effect in the results of the examination of the urine of epileptics immediately after a fit, by Huppert (*Virchow's Archiv*, vol. lix.) and others, who found albumen in many such cases; but the peculiarity here is that the *quantity of albumen is always trifling*, unless the cases are complicated with renal disease; whereas the albuminuria of the puerperal state, at the period of convulsion at least, is very marked. But it is not impossible therefore for the milder cases of puerperal eclampsia due to reflex irritation thus to acquire a small albuminuria during the convulsions where previously none was present. Against this we should, however, guard. But the very circumstance makes it likely that a large albuminuria is due to a more decided alteration of the structure of the kidney than the mere congestive condition which is the cause of a small one.

Now as to the *cause of these renal changes* which lie at the bottom of so large a number of cases of puerperal eclampsia. How are they induced? Two views are held, by as many sets of observers, as will be recalled from the abstracts presented. One, held by Frerichs, Braun, Litzmann, Scanzoni, and others, attributes them altogether to pressure upon the emulgent veins, caused by the pressure of the pregnant uterus, producing congestion, albuminuria, and imperfect elimination of matters usually thrown off by the kidneys. This view is favored by the fact that albuminuria and eclampsia are most common in primiparæ, and in multiparæ with twin pregnancies, hydramnos, deformed pelvis, or other condition which increases the intra-abdominal pressure. On the other hand, it is objected to this view that albuminuria very early in pregnancies, some instances of which have been observed within two months after conception, cannot be thus accounted for. And it will be recollected that Bartels, in his article on the "Parenchymatous Nephritis of Pregnancy," in Ziemssen's *Cyclopædia of Medicine*, attempts to prove the anatomical impossibility of such pressure, while he claims also that congestion of this kind should be followed by interstitial nephritis instead of catarrhal nephritis, the conditions being similar to those in mitral disease of the heart, in which, from venous

pressure, the blood is backed into the kidney and liver, in both of which is found the hard kidney of an interstitial nephritis.

Those who believe in the pressure theory attribute the albuminurias of early pregnancy to pre-existing renal disease,—which is not impossible.

The opposite party ascribe the parenchymatous nephritis to an intoxication of the blood, due to the increased amount of excrementitious matter which must enter it from the retrograde metamorphosis of the tissues of the fœtus, as well as of the mother; and consider its operation to be like that of the poison of scarlatina, which similarly induces a catarrhal nephritis. The reasons for this view are well given by Dr. Peter in the lecture already quoted, and I need only refer you to them.

I think the mistake consists in the adoption of either view to the exclusion of the other. Doubtless both contribute to it in varying proportions, according as circumstances favor the operation of one or the other. Certainly the greater frequency of the eclampsia in primiparæ, and the other situations above named, can only be accounted for on the pressure theory; while the force of Bartels's argument cannot be denied.

It must not be forgotten, as stated at the outset, that the pregnant woman is liable to acquire Bright's disease from the ordinary exciting causes of renal diseases,—exposure to cold and wet, excessive eating and drinking, or the absorption of some zymotic blood-poison; or even from absorption of septic matters, possibly from a putrid fœtus or morbid material from the interior of a partially-contracted uterus. These causes are all well tabulated by Dr. Johnson in the extract from his lectures.

Now, as to the toxic agent itself in those cases of puerperal convulsions due to Bright's disease, I doubt whether it comes properly within my task to discuss it to-night. But a very few words will dispose of it. It is very true that in a few instances only has an excess of urea been demonstrated in the blood of puerperal eclamptics. It has, however, been unmistakably shown to be present in some. Frerichs, it is well known, sought to prove that it was not urea, but carbonate of ammonium into which the urea was converted, and both Prof. Hammond, of this country, and Dr. B. W. Richardson, of London, have ably refuted it, although Spiegelberg has recently reasserted the original view of Frerichs, based on a series of experiments conducted by himself and Heidenhain (London *Lancet*, 1870). Hammond and others have also proven that the urine itself is a much more efficient agent in producing the symptoms of the so-called uræmia; and there is little doubt in my mind that it is not urea, or carbonate of ammonium, or any single substance, but it is the entire mass of excrementitious

substances usually eliminated by the kidneys which, retained in the blood, give rise to the uræmic symptoms of Bright's disease, and of those cases of puerperal eclampsia depending on Bright's disease.

If it be asked how it happens, on the supposition of so grave a disease as parenchymatous nephritis, that recovery is ordinarily so rapid when labor has successfully terminated, the answer is again easy. Let it be remembered that we have ordinarily a case of catarrhal nephritis of short duration intensified by embarrassed circulation, if not primarily caused by it. This cause removed, the blood moves freely, the kidneys act rapidly, the quantity of urine is largely increased, and with it the excrementitious matter. The convalescence is therefore rapid. It is, however, no less so, and the case differs, indeed, in no way from that in catarrhal nephritis after scarlatina, where the toxic agent has not operated for so long a time or so virulently as to produce chronic changes. And it is well known that the earlier the symptoms appear, and the longer the disease has lasted before relief comes, and therefore the more deep-seated the lesions, the longer the albuminuria and the casts continue afterwards; while in a certain class of cases these symptoms continue, the relief does not come, the disease becomes chronic.

In the discussion which followed the reading of the paper, the following remarks were made:

Dr. Albert H. Smith said he fully agreed with the views expressed in the paper and the conclusions arrived at; even going further than the author, in stating that, so far as his experience had gone, he felt justified in adopting the belief that all *genuine* puerperal convulsions are the result of renal disease. He would divide the convulsive seizures arising in pregnancy into two classes,—those from hysterical and those from renal causes; the former so easily distinguished from the latter in the character of the spasmodic movements in the facial expression, and in the partial consciousness and appreciation of surrounding conditions, as to leave no question as to their true nature. Convulsions arising from brain-hyperæmia or from brain-anæmia he had never seen as attendant upon labor. In every case of true puerperal convulsions he had found great quantities of albumen, and in all cases examined microscopically casts were present.

The theory of the albumen in the urine found at the time of convulsions, and not recognized previously, being the result of the brain disturbance, was hardly tenable, when we consider the great amount of albumen found in these very cases of sudden appearance, the urine in many instances solidifying under heat, so that a test-tube or spoon in which it is heated

may be overturned without disturbing it, and in malignant and fatal cases profuse hæmaturia being present. We have all of us seen albumen in the urine detected after convulsion from brain-tumor, epilepsy, or other central conditions, but it is in quantities so small as to require delicate tests to discover it; Heller's test, for instance, giving a faint zone only at the margin of the acid.

The theory of anæmic origin, so strongly urged some years ago in a review in *Hays's Journal*, by the late Prof. Carson, is from an experimental point of view equally untenable. If the condition of anæmia bore any relation to convulsions as cause to effect, then, at any rate, we ought to have convulsions occurring after flooding; for if a slowly-developing anæmia, to which the brain would gradually accustom itself, would be a cause of eclampsia, how much more would the sudden abstraction of blood be attended with violent convulsions! But so far experience does not sustain this position that no relation between the two is ever seen in obstetric practice.

Dr. Smith had seen a vast number of cases of hemorrhage, both from placenta prævia, occurring weeks before labor, and from flooding after labor, and had seen the subjects of these conditions as nearly moribund from loss of blood as could be to live, pulseless, and with respiration almost suspended, and yet never had he seen puerperal eclampsia following such a case.

That the condition of pregnancy in itself predisposes to renal disease cannot be questioned; why or how, cannot be explained. But it is a matter of common experience that in cases of chronic albuminuria, when, under treatment, a diminution of the renal symptoms has taken place, the occurrence of pregnancy kindles up the spark into an active flame, and the chemical and microscopical tests and the physical and rational signs all show a marked and steady increase of the kidney-trouble. Dr. Smith has had a patient for over nine years with chronic Bright's disease, in whose case treatment temporarily subdues the condition; but so rapidly does pregnancy aggravate it that on two occasions the examination of the urine, showing a great increase of albumen, has enabled him to diagnosticate pregnancy before the suppression of the menses had led the patient herself to suspect it. But in addition to this tendency to albuminuria from the mere condition of the pregnant woman, we have a powerfully-exciting cause in the pressure of the gravid uterus upon the renal vessels, more apt, as Dr. Tyson has shown, to occur in primiparæ; also, as is seen, in women having a long rest between their pregnancies, in women with multiple pregnancies, and with pressure from the distention of a uterus with

amniotic dropsy. And it is in these cases in which the development of the renal symptoms is rapid that we have the greatest tendency to convulsions and the most violent and fatal form of them when they arise.

Dr. Smith agreed with Scanzoni, that the more decided the evidences of renal disease, the more violent the convulsions and the more apt to be fatal. Recovery after the appearance of marked hæmaturia, with urine solidifying under heat, is the exception, and yet in cases in which the urine becomes albuminous early in pregnancy, and continues slowly to increase in this condition, we rarely have convulsions, unless set up by a sudden aggravation of the kidney-lesion by some rapidly-developing cause. In this respect there is a resemblance to the history of the albuminuria of scarlatina, in which we observe that in those cases in which the renal trouble shows itself early, and proceeds by a steady course, we very rarely have convulsions; whereas in cases suddenly becoming œdematous, and showing the presence of large quantities of albumen, from exposure to cold, we very frequently have those symptoms culminating in eclampsia. And we have in the scarlatinal uræmia, in its complete history, a very strong argument by analogy in favor of the uræmic origin of puerperal convulsions, based upon the precise identity in the character of the convulsive movements, in the mode of onset, and the conditions of brain following each spasm, as well as those resulting from a continued repetition of them. The fact already mentioned, of convulsions having their violence and fatality proportioned so fully to the rapidity of development of the renal disease, observed as common to both affections, is further sustained as an argument by another equally well known fact, that often the first condition of things to call attention to the kidneys is the simultaneous appearance of anasarca and convulsions in patients previously considered in good health, the one in a normal physiological state of pregnancy, the other as fully convalescent from a mild attack of fever. And as a further point in the analogy, we often make in both conditions, even when anasarca and the general evidence of renal troubles are present, fruitless examinations of the urine for the discovery of either albumen or casts; finding at one time such conditions, while at others they are entirely absent and yet without any change in the general symptoms. But no one would consider this as militating against the theory of scarlatinal eclampsia being renal in its origin: why, then, should the fact that some observers have at times failed to find albumen in the urine of patients, *before* labor, who had eclampsia, followed by the *detection* of albumen during labor, be looked upon as a conclusive argument against the renal origin of puerperal convulsions?

Dr. Fred. P. Henry said that Dr. Tyson's paper had confirmed his

belief that Bright's disease is more than usually malignant when associated with the puerperal state. Under the term malignant he referred particularly to the explosive symptoms, coma and convulsions. This fact acquires a special interest when it is pointed out that there are conditions under which Bright's disease is rendered peculiarly *benign*. On a recent occasion he had endeavored to show that Bright's disease is very favorably modified as to its explosive symptoms by its association with phthisis. At that time he had seen several cases whose study had induced him to advocate this view, and since then he had seen several more. Patients with great œdema, albumen, and casts in the urine and cavities in the lungs, have retained their consciousness until almost the last moment of life, and have had no convulsions. Any one of large clinical experience must have observed this fact. Dr. Henry had endeavored to account for it by the insufficient oxidation of the blood due to the lung-disease. It is well known that Frerichs advocated the view that it is not the urea that causes the explosive symptoms, but carbonate of ammonium, a substance containing an additional atom of oxygen. Whether this be so or not, Dr. Henry is of opinion that diseases attended with insufficient oxidation of the blood, such as phthisis, modify Bright's disease in a favorable manner as regards the explosive symptoms. On the other hand, during gestation these processes of oxygenation are very active. Oxygen is required both for the mother and fœtus, and during the active interchange of gases the accumulated urea is readily converted into a more poisonous substance, whether this be carbonate of ammonium or some other.

It is customary to speak of the puerperal period as one of hydræmia, and this view was ably advocated by the late Prof. Carson; but such opinions have generally been founded upon theoretical considerations and the gross appearances of the blood. Even the microscope cannot determine absolutely the condition of the blood; a minute examination with Malassez's apparatus will, of course, give the relative richness in cells, but will not give any idea of the total amount of blood in the body, which certainly has a bearing upon the question.

In regard to the mechanical theory of pressure as a cause of Bright's disease during gestation, by impeding the flow of blood through the emulgent veins, it would require to be shown, in order to sustain it, that the forms of kidney-disease associated with diseases of the heart, impeding the return flow of blood, are identical with the forms associated with pregnancy.

Dr. C. B. Nancrede said he did not think Bright's disease, occurring in the puerperal state, could be due to pressure, since we have it coming on early in pregnancy; nor is one kidney more affected than the other. The

form of the disease is not that which is caused by obstructions, which is brown induration. The uterus cannot press upon the emulgent vein, since we have a large amount of intestines between the two. In the cases of tumors of the uterus we do not have this disease, according to most authorities; again, we have it in multiparæ. We meet with it much more frequently in twin pregnancies, even where the element of pressure is largely eliminated.

Dr. H. Lenox Hodge said that in distentions of the abdomen from ovarian tumors, albumen is sometimes found in the urine. It seems, in these cases, to be due simply to pressure. Although the pressure may be very great, and albumen present in the urine, yet convulsions do not occur. He had never known a case of convulsions due to the pressure of an ovarian tumor. Pressure does not seem enough to account for convulsions. In pregnancy there is an unusual nervous excitability, and the blood is filled with the effete products from two beings, in addition to pressure. Those who thought that in pregnancy there was no pressure on the renal veins appear to have overlooked the fact that the pressure is caused not by the direct contact of the uterus, but by the coils of intestines filled with gas and liquid, which like air-cushions transmit pressure in every direction, around every curve and in every notch.

Much light in many disorders is thrown upon their pathology by the results of the treatment adopted, and by the subsequent history of the case. In the treatment of puerperal convulsions, free venesection is recommended by the majority of authorities, although bleeding has fallen into disuse in most other disorders. The success of those who bled freely in puerperal convulsions has been very marked. Dr. Tyson alluded to the experience of Dr. Hiram Corson as being greatly in its favor. Dr. Hodge said that his father could not recall a single case of puerperal convulsions which he had lost when he had charge of it from the beginning. He bled, and bled freely. On the other hand, a very different treatment is followed in Bright's disease, and certainly very few would dare to bleed.

Then, again, as regards the subsequent history of the case. In Bright's disease there is a history of progressive danger. It generally ends in death. In the most favorable cases, if recovery takes place, there is a tedious convalescence. In puerperal convulsions the danger is limited to pregnancy, to labor, and to a little while after labor. If this period be passed, no matter how severe the convulsions have been, there is rapid improvement and rapid restoration to health. Such different results must be due to some difference in pathological condition.

Dr. Tyson said he had already referred in his concluding remarks to the two views now held; one ascribing the result to a congestion due to pressure by the gravid uterus on the renal veins, the other to a poison accumulating in the blood, similar to that of scarlatina, and acting similarly; that against the former it is urged that abdominal tumors of equal size with the gravid uterus do not cause albuminuria; that Bright's disease sometimes occurs in the earliest months of pregnancy, when it is impossible that pressure should be exerted by the gravid uterus on the renal veins. That against the latter it is urged that, except in cases where the Bright's disease pre-existed, it very seldom originates in multiparæ, but only in primiparæ, and in multiparæ with plural births where pressure is most likely to be exerted. But since, as stated by Dr. Hodge, cases do thus occasionally occur early in pregnancy, and albuminuria is sometimes found caused by the pressure of large abdominal tumors, we must, in truth, admit the occasional operation of both causes; although he believed the second was the more frequent, especially as the form of kidney-disease almost invariably found is the catarrhal, or tubal nephritis; whereas the form found in congestion of the kidney due to obstruction is always interstitial nephritis, in which the connective tissue is the seat of proliferation.

As to the prognosis of the two conditions, scarlatinal nephritis and the nephritis of pregnancy, he had also pointed out a similarity. If the poisoning is not too profound, and no lesion results from the convulsion itself, or the kidney is not permanently damaged, recovery is likely to take place in both. And as to the therapeutics being radically different in the two cases, Dr. T. was by no means certain that bleeding would not be as good a remedy in the convulsions of acute Bright's disease as in puerperal eclampsia, and, although it is never practised, the treatment which is found to be of most service under these circumstances—*active purgation, sweating*, etc.—is of the same character as bleeding, and operates in the same general way. Bleeding might be of great advantage in the convulsions of Bright's disease, and his friend Dr. Hiram Corson had already been referred to (in the body of the paper) as having used it with advantage. The difference in the two conditions lies chiefly in this, that the kidney is generally less damaged in the puerperal condition, and, having the cause removed, the organ is enabled to return rapidly to its original state and perform its function of elimination; while in the scarlatinal nephritis sometimes, although not always, the poison has operated to such a degree as to produce permanent organic change in the organ, which cannot be removed, and thus the acute condition becomes a chronic one.

April 25th, 1878.

2. *Biceps muscle filled with trichinæ.*

Presented by Dr. J. G. Richardson.

I regret that the history of this case is so incomplete, although full enough to illustrate an important point in regard to trichiniasis, namely, the frequency with which it escapes detection.

The body from which this biceps muscle was removed was a subject upon one of the tables in the anatomical rooms of the University of Pennsylvania. In the course of their dissection, one of the class of students engaged upon it observed a peculiar dotted appearance in the pectoral muscles, and, calling the attention of Prof. Rothrock and Dr. B. F. Lautenbach to this condition, they at once discovered the parasite. No account of the case could be collected, but the man, apparently a German of some 40 or 50 years of age, well nourished, and the victim of an accident, had been subjected to a *post-mortem* examination, in which it is fair to presume that the trichinæ had not been detected. This shows that an autopsy does not always suffice to demonstrate the existence of trichiniasis, and suggests that a more thorough examination, including a microscopical one, of the muscles, especially of the pectoralis major, should *always* be made.

The parasites are here encysted in calcified cysts, and as this does not generally occur in less than two years, we may conclude that the onset of the malady antedated that period, and may of course have preceded it by fifteen or twenty years.

This example of trichiniasis is, I believe, only the fifth that has been observed in Philadelphia, and it is a curious coincidence that in lecturing upon the prophylaxis of this malady in my course on Hygiene, last spring, I urged our students almost exactly one year since to examine most carefully all the subjects upon the dissecting-room tables, as I thought it likely that cases of trichina disease not infrequently occurred and escaped detection simply through want of scrutinizing observation. It also seems probable, as I then suggested, that the presence of these parasites in their encysted form may be a more or less direct cause of some chronic disturbance of function or nutrition as yet unrecognized.

May 9th, 1878.

3. *Case of disseminated miliary tuberculosis.*

Presented by Dr. Wm. Pepper.

H., æt. 34, clerk in Pennsylvania Railroad office. Saw him May 5th, 1878, with Dr. Stillwell. Married six years. During the honeymoon became insane, and remained so six months. Always correct in habits, so far as known. No history of syphilis. Again was insane, two years after, for four months. Also, at least a dozen times, while sitting conversing, would suddenly jump up, say his head ached violently, become insane, continue so for fifteen to forty minutes, fall into deep sleep for twelve or eighteen hours, and awake weak but rational. *Was this epileptic?* About December 1st, 1877, began to cough, and lost flesh; and by April 23d, 1878, when he consulted Dr. S., marked symptoms of disseminated tubercle of left lung. By May 1st, 1878, some signs of mental disturbance; headache; drowsiness; pulse irregular; loss of appetite.

May 5th, 1878, almost unconscious. No squint. No vomiting. Belly normal. Tâche méningitique quite marked. Pulse regular, 118. Respiration regular and rapid. Physical signs as before,—extensive disease of left lung and of right apex.

May 8th, 1878. Symptoms advanced; squint; eyes glazing. Pulse 148. Respiration abdominal, very rapid. No tidal breathing observed, but it may have existed, as I saw him only on May 5th, 6th, and 8th, 1878. Died same day.

Post-mortem.—Skull normal. Brain congested. Lateral ventricles contained a great deal of turbid serum. Walls very much softened. Great deal of lymph. Imbedded miliary tubercles at fissure of Sylvius on both sides over pons, optic chiasm, and intervening parts of base.

Lungs.—Left riddled with tubercles, gray and yellow; in places disseminated, in others confluent. A few cavities. Right lung, scattered miliary tubercles. Strong pleural adhesion on left side. Disease evidently quite old there. No pericarditis.

Liver congested; thickly studded with miliary tubercles.

Spleen enlarged. Capsule thickened, and patches of lymph. Numerous miliary tubercles in tissue.

Kidney congested. Quite numerous gray tubercles, larger than in spleen.

No peritonitis. Mesenteric glands not enlarged.

Microscopical examination of brain, kidney, liver, and spleen.

By Drs. Formad and Gilliard.

Kidney.—Was found to present a positive increase of interstitial connective tissue corresponding in appearance to the early stage of interstitial nephritis. The epithelial cells, in places, were in a state of cloudy swelling, in other places granular, and in still other spots had undergone complete fatty change.

Some of the uriniferous tubules were filled with glistening hyaline casts.

Disseminated throughout the parenchyma of the kidney were found well-defined tubercles, which were so large as to be easily recognized in a thin section by the naked eye. They presented the usual reticulated arrangement of adenoid tissue filled with more than the common number of lymph-corpuscles. In places the tubercles showed spots of secondary cheesy softening; in other places they had undergone complete cheesy change.

The tubercles have evidently originated in the capillary plexus making up the Malpighian tufts.

Liver.—Shows a large number of tubercles of large size, situated principally in the outer zones of the acini; some isolated; some aggregated in groups of from four to six; many of them characterized by the presence of the so-called giant cells; some have undergone cheesy change. There are also seen isolated spots of fatty infiltrations. Otherwise the parenchyma of the liver seems not to be impaired, except that the cells, more especially those of the outer zones of the acini, are stained with bile.

In some places between the acini are seen bands of adenoid tissue.

Spleen.—There are a great many tubercles along the smaller vessels, some isolated, others in groups.

Brain.—Pia mater.—All the blood-vessels, as well as the capillaries, are covered with tubercular masses, in many places entirely hiding the muscular structure, and in some points aggregated, forming spindle-shaped or round nodules, entirely surrounding the vessel, in other places forming a bulging on one side.

Portions of brain-tissue from the neighborhood of the fissure of Sylvius and from the ventricles showed partial softening, as indicated by the presence of compound granular cells, pus-corpuscles, and the débris of nerve-fibres. Other portions appeared to be normal, but the vessels throughout, capillaries and blood-vessels, were surrounded and covered with tubercular masses aggregated in places to form nodules. *May 29th*, 1878.

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